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I

WHAT IS PERCEPTION DEAFNESS FROM A PHYSIOLOGICAL AND HISTOLOGICAL BASIS?*

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BOSTON

To discuss all of the physiology of hearing from the histological structures involved in this sensory mechanism is not possible. One must limit oneself to some of the facts that have been discovered in the past few years and correlate them with histological structures present in the organ of hearing.

Hearing is not, as most of us think, a process that concerns the ear alone. What one hears is the interpretation by the brain of nerve messages or impulses sent to it by the sense organ of sound (organ of Corti). Hearing, as sight or any of the other special senses, is a mental phenomenon, and is, therefore, an intimate and personal experience. Though the perception organ for sound (organ of Corti) is the same for all mammals, the interpretation of the stimuli received depends on the mental capacity of the animal or human being to interpret and make use of these stimuli. (Figs. 1 and 2.)

*Presented at the Meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, Mass., January 11th, 1939.

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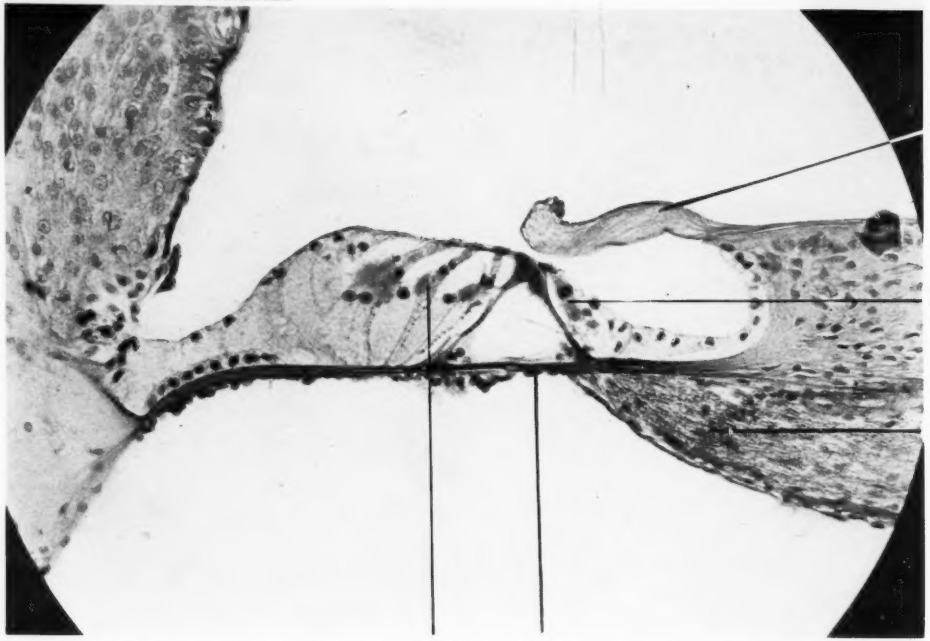
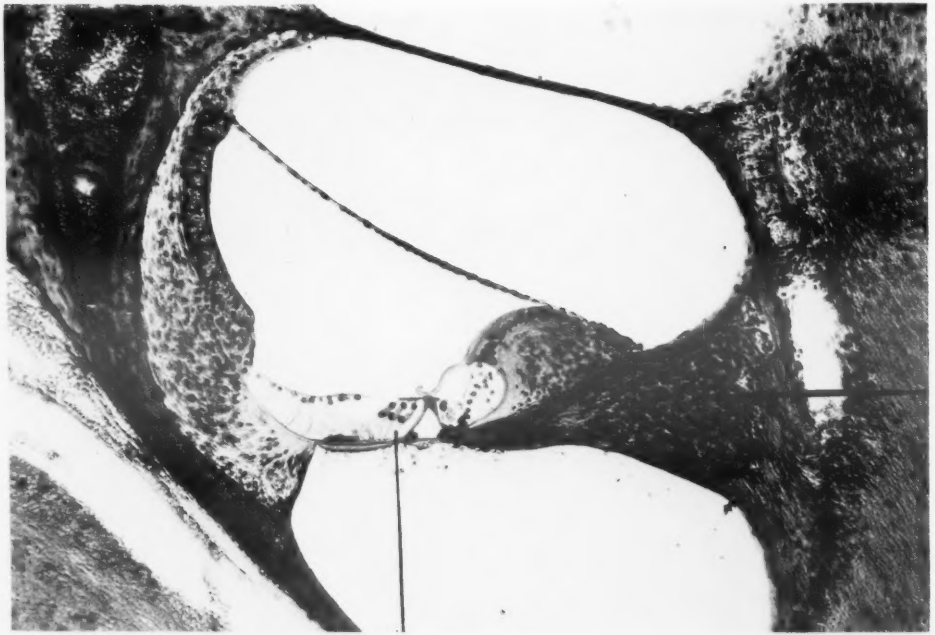


I.H.C.—Internal Hair Cell
B.M.—Basilar Membrane
C.N.—Cochlear Nerve
E.H.C.—External Hair Cell
T.M.—Tectorial Membrane

Fig. 1. Normal organ of Corti, dog. Photomicrograph. Note the marked similarity with the human organ of Corti. The internal hair cell and internal pillar cell rest on bone. The external hair cells on the basilar membrane.



O.C.—Organ of Corti

S.G.—Spiral Ganglion

Fig. 2. Cochlea, organ of Corti, mouse. Photomicrograph. Normal organ of Corti of mouse. Compare with normal organ of Corti of dog.

Deafness, or the inability to hear, thus divides itself into two phases:

1. The inability of sound to reach the sense organ, or, as we know it, the middle ear or conduction deafness.

2. The inability of the end-organ or brain to receive and interpret the sound waves—perception or nerve deafness.

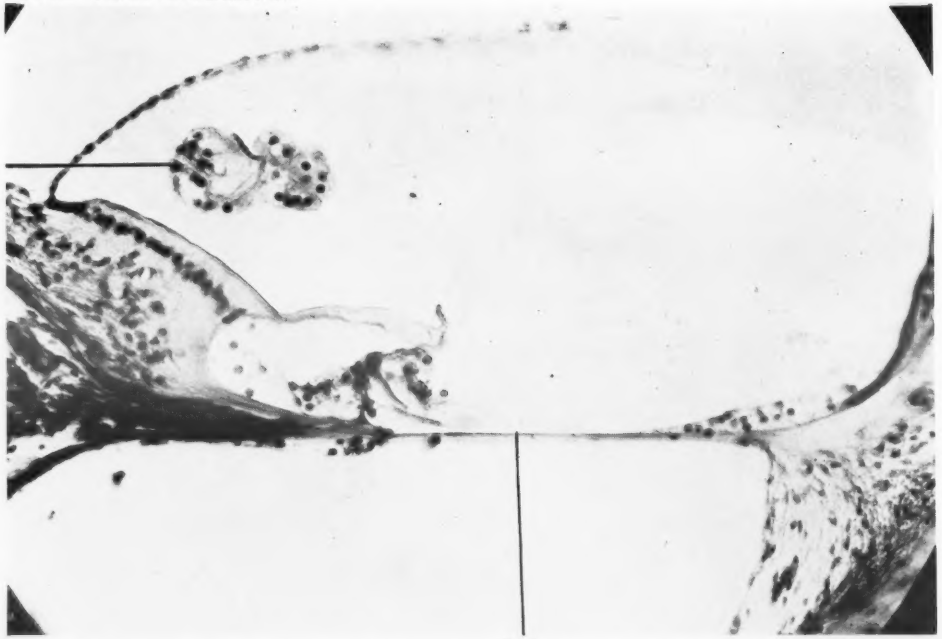
In this discussion conduction deafness will not be considered, for, in its final analysis, it is a mechanical deafness rather than a true deafness.

Perception deafness is the result of the inability of an individual to interpret the sounds heard into intelligent language. How often do we hear the deaf patient say, "I hear the sounds but cannot understand what you are saying." In these cases the evidence points not to the brain's inability to interpret the sounds but to a defect in the perception and the transformation of sound into accurate nerve impulses.

Thus one must study the changes that occur in the perception organ to explain this type of deafness. The neuroepithelium of the perception organ is the organ of Corti. This structure histologically was first described by Corti in 1851, and from his day to the present numerous details have been added to the histology and neurology of this organ.

The normal cochlea with its organ of Corti does not need to be described in detail, as it can be found in most elementary text-books on histology or physiology. But there are some facts about the organ of Corti that have not been appreciated.

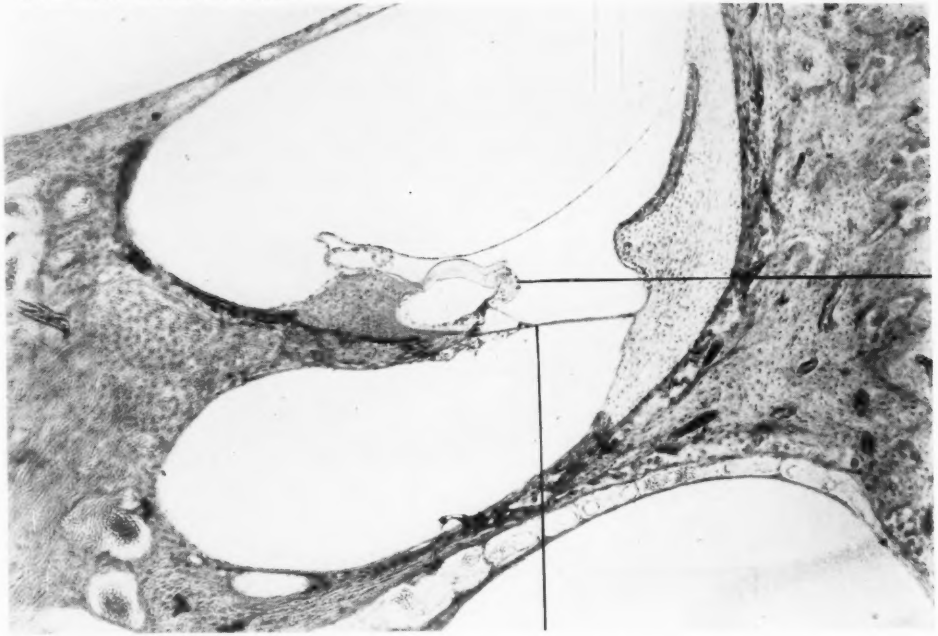
The tectorial membrane should be regarded as a modified otolithic membrane. Its function is similar to the otolithic membrane of the vestibular apparatus, and it is a structure which aids in the movement or bending of the hairs in the external and internal hair cells. Wittmaack[7] recently claimed that the hairs of these cells are artefacts. The evidence for this is rather slight and can be disregarded, for one must remember that the organ of Corti is essentially an organ of touch and has developed from the embryonic skin. One can call attention to the fact that even in the eye the rods and cones have a modified hair process, for they also have developed from the neuroepithelium of the embryonic skin. The tectorial membrane is in intimate contact with the organ of Corti. Its usual appearance in histological section is the result of fixation.



E.H.C.—External Hair Cells

B.M.—Basilar Membrane

Fig. 3. Organ of Corti, guinea pig. Photomicrograph. Organ of Corti thrown off the basilar membrane by a sudden intense sound. The basilar membrane and Reissner's membrane are not ruptured. Note that the external hair cells are floating free in the scala media.



E.H.C.—External Hair Cells

B.M.—Basilar Membrane

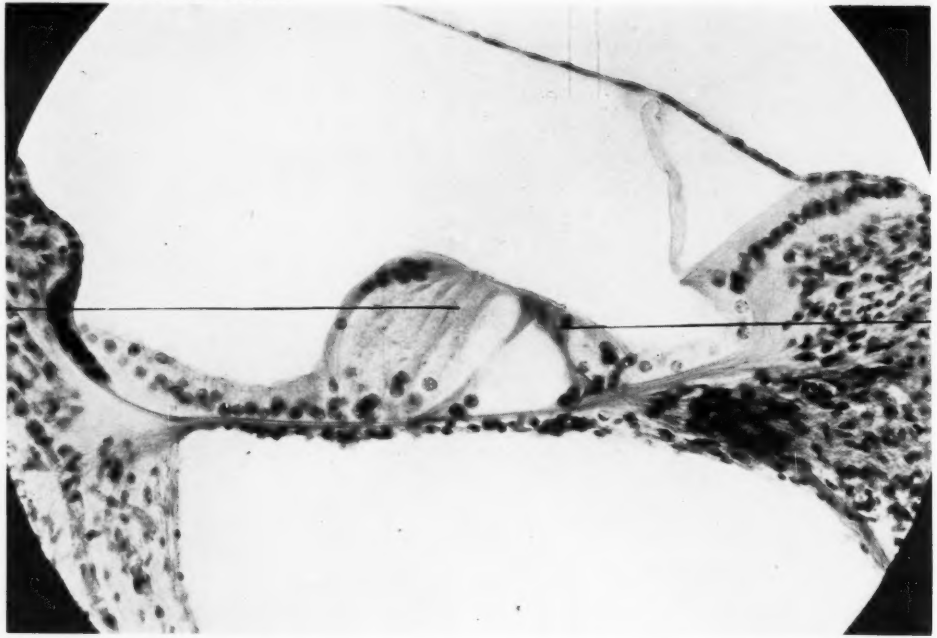
Fig. 4. Cochlea, cat. Photomicrograph. Organ of Corti thrown off by intense sound of 400. Note that only the external hair cells and supporting cells have been thrown off.

The stria vascularis, from its structure, is similar to the choroid plexus of the brain, and the endolymph is secreted by it. That changes in the stria vascularis can cause deafness is questionable, for in many animals with normal hearing that were studied, changes have been found in the stria vascularis.

The basilar membrane, on which the organ of Corti rests, has been the center of attack since the day of Helmholtz (1860), who first developed the Harp theory (resonance) on the basis of difference in width of the basilar membrane. The question has been raised: Is vibration of the basilar membrane necessary for hearing? Shambaugh[6] has maintained that it is not necessary. He has shown the portion of the basilar membrane near the round window to be actually osseous with the organ of Corti resting on it, but there has been no proof offered that the animal heard with this small portion of organ of Corti. Recent experiments[4, 5] have shown that the basilar membrane can be made to vibrate to such a degree that the organ of Corti is thrown off the basilar membrane, with a resulting loss of response for this region. (Figs. 3 and 4.)

The organ of Corti only rests partially on the basilar membrane. The external hair cells and the external pillar cell rest on the membrane. The internal hair cell and the inner pillar cell rest on bone. This allows for greater movement of the external hair cells and pillar cell than for the internal pillar cell and internal hair cell. The internal pillar cell has a sort of hinge socket for the head of the external pillar cell. The significance of these facts becomes evident in the study of the function of the external and internal hair cells. (Fig. 1.)

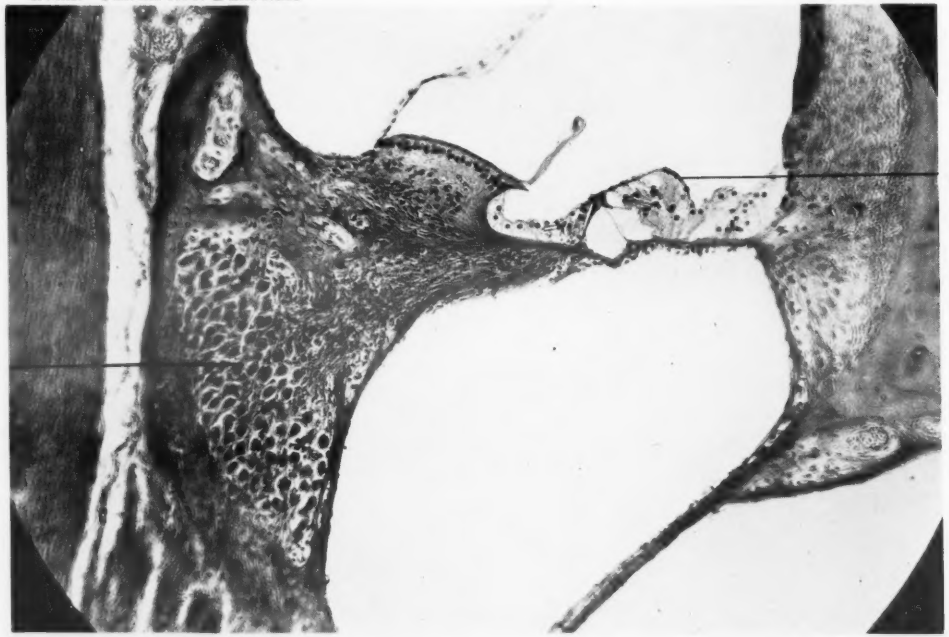
The external hair cells lying on a movable membrane are of necessity subject to greater stimulation than the internal hair cells, for it will take very small movement of the basilar membrane to excite these cells, while the internal hair cells resting on bone can be stimulated only when the push or pull of the basilar membrane causes a far greater distortion of the organ of Corti. If this assumption is true, then experiments causing the basilar membrane to be vibrated for long periods of time should cause changes in the external hair cells before they are evident in the internal hair cells. In animals (guinea pigs) exposed for a long period of time to an interrupted tone of 2,500 cycles at high intensities this was found to be true.[4, 5] The external hair cells had degenerated before the internal hair cells. Accompanying this degeneration of external hair cells was a loss of response from the organ of Corti by the Wever and Bray



E.H.C.—External Hair Cells

I.H.C.—Internal Hair Cell

Fig. 5. Organ of Corti, guinea pig. Photomicrograph. Organ of Corti of animal exposed to interrupted intense sound for a month. Showing a degeneration of the external hair cells. Note the internal hair cell is normal with a normal nucleus.



S.G.—Spiral Ganglia

O.C.—Organ of Corti

Fig. 6. Cochlea, guinea pig. Photomicrograph. Normal side of guinea pig with eighth nerve cut on opposite side.

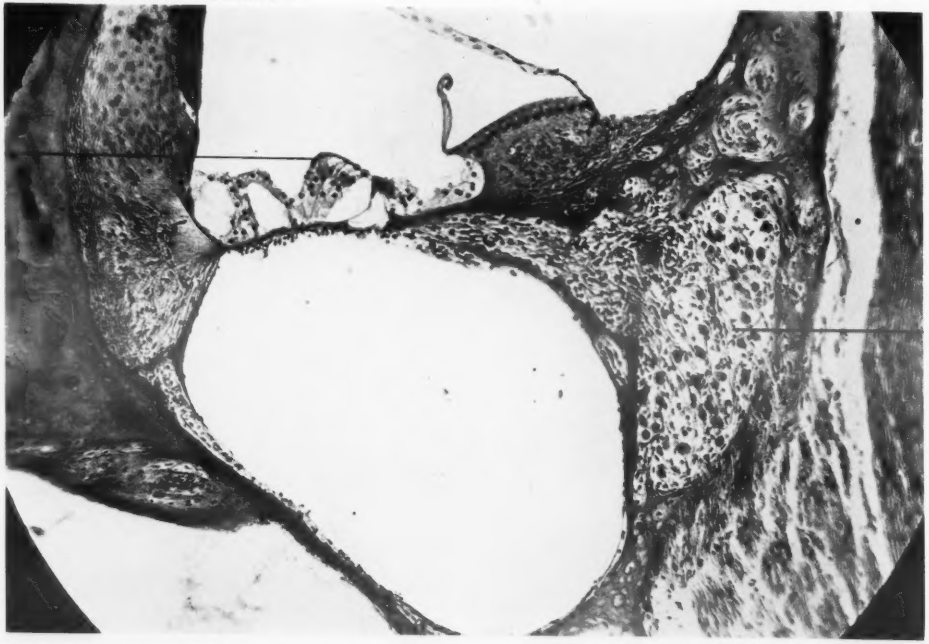
method from 10 to 40 decibels, depending on the number of external hair cells destroyed. In fact, the loss of hearing was very typical of boiler makers' deafness. Thus, by excessive mechanical stimulation, a degeneration of external hair cells was obtained. Here it must be noted that the animals were not totally deaf but lost only part of their hearing, the internal hair cells still responding to sounds but at higher intensities. These experiments indicated a difference in function between the two types of cells. The external hair cells respond to stimuli of a weak nature. The internal hair cells respond to those of greater intensity. The internal hair cells beginning to function, it is thought, at intensities between 30 to 40 decibels above the threshold. (Fig. 5.)

Other animals with pathological changes in the organ of Corti from unknown causes also confirmed these facts.[4, 5] Several animals were found to have degeneration of the external hair cells throughout the organ of Corti with a loss of 30 to 40 decibels throughout the range.

Animals made deaf by use of quinin also showed similar degeneration of the external hair cells. This means that the external hairs are not only more sensitive than the internal hair cells, but also more liable to degeneration, either from trauma (excessive noise) or toxic poisoning by drugs or disease.[4, 5]

Perception deafness then can be considered as a degeneration of the external hair cells in the organ of Corti, a scattered degeneration causing slight losses, a more general degeneration greater losses, and complete loss of hearing with degeneration of both internal and external hair cells. Crowe and Guild[1] have shown that in the human with high tone loss there is a loss of hair cells at the basal portion of the organ of Corti. There is also a difference between the internal and external hair cells histologically, the internal hair cell being a larger cell in comparison with the external hair cell. The external hair cell is more slender and longer.

The nerve supply of the organ of Corti also furnishes evidences of a difference in function of these two special types of hair cells. A single nerve innervates one or two internal hair cells at the most, while a nerve to the external hair cells will be in contact with a large number, Lorente de No[3] having traced these nerves for a quarter of a turn in the cochlea. Thus we have a large number of external hair cells supplied by a single fibre, and only one or two internal hair cells by a single fibre. This points to the fact that



O.C.—Organ of Cort

S.G.—Spiral Ganglia

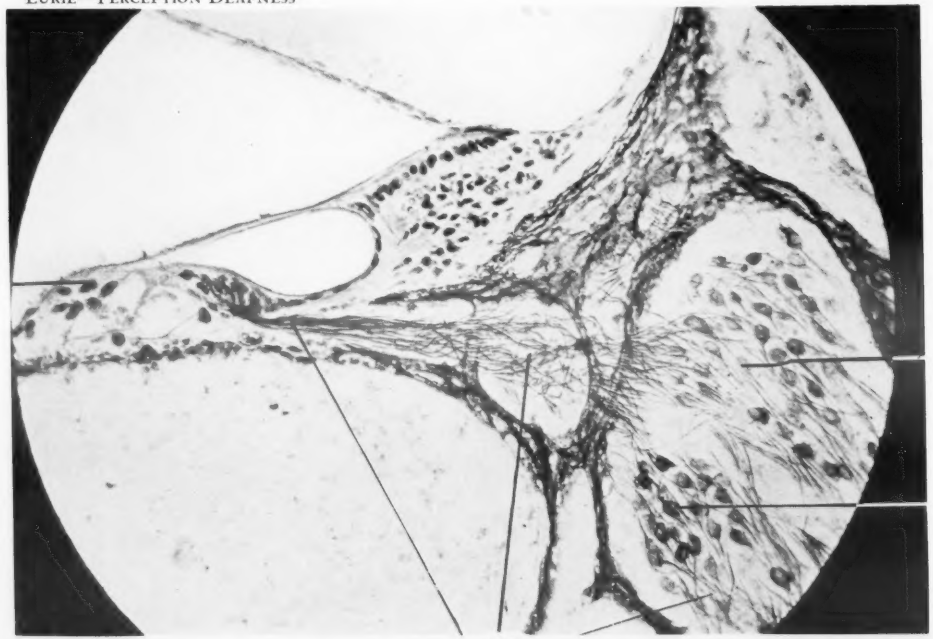
Fig. 7. Cochlea, guinea pig. Photomicrograph. Eighth nerve cut on this side. Note degeneration of the ganglia cells, and normal organ of Corti present.

sounds of small intensities moving along the basilar membrane will be picked up by the external hair cells. For, with a multiple nerve supply, these slight changes can be detected more easily. There is also no fine discrimination of pitch with the external hair cells. The single nerve supply of the internal hair cells points to a more accurate discrimination of pitch, since a difference could be detected two internal hair cells apart. With the external hair cells this difference must be many cells apart. This has been found true by psychological tests on the discrimination of pitch at various intensities, which have shown that the discrimination of pitch at intensities below 30 decibels were about 500 compared with 1,500 different pitches determined at intensity levels above 60 decibels.[5]

The cochlear nerve is a bi-polar sensory nerve and is no different than any other post ganglionic nerve (Fig. 8). The ganglion cells themselves are large, though not as large as the vestibular ganglion cells. With degeneration of the ganglia or of the nerve, deafness results, but this type of deafness is of a complete type whether for the whole range or part of the hearing range.[1] The organ of Corti can remain functioning for a long period of time after the cochlear nerve has been damaged. Experiments in which the cochlear nerve[2] was cut showed a normal appearing organ of Corti with normal response by the Wever and Bray method three months after operation. The ability of the end-organ to survive nerve damage for considerable time may explain the recovery of hearing in cases that have had complete deafness during the course of a disease. Here the lesion is of the nerve itself, and when regeneration occurs or the neuritis disappears, hearing again is recovered, for normal connections are thus re-established between the organ of Corti and the central nervous system. (Figs. 6 and 7.)

There are other physiological facts of hearing, such as masking and loudness, that also can be correlated and explained from the nerve supply of the organ of Corti.

The organ of Corti is an organ for the perception and analysis of the sounds received by it. It is as intricate in this process as the eye is in perceiving and analyzing color. With our present knowledge of the cochlear apparatus we can explain a number of the phenomena, but the great mystery in the ear is still how we hear a variety of sounds at one time and keep them distinct and separate. This phenomenon at the present time defies explanation and is similar to the eye seeing many objects and colors all at the same time and



N.F.—Nerve Fibres

G.C.—Ganglia Cells

O.C.—Organ of Corti

S.G.—Spiral Ganglia

Fig. 8. Cochlea, guinea pig. Photomicrograph. Bodian nerve stain showing the nerves of the organ of Corti going to spiral ganglia. The ganglia cells are bi-polar, similar to the spinal ganglia of the spinal nerves.

in their proper relationship. For the eye and the ear act alike in a great many ways.

Thus there are a few basic facts that can be explained by the histological structure of the organ of Corti. At the present time there is so little we know of how these cells and their nerves function, that only with the further discovery of fundamental phenomena associated with neuro-sensory cell activity will further progress be made.

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II

SULFANILAMIDE IN OTOLARYNGOLOGY: SOME PERSONAL EXPERIENCES*

RALPH A. FENTON, M.D.

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Hundreds of papers respecting sulfanilamide and its therapy have appeared in the past year, and in our field important recent contributions have been made by McMahon,[1] Kopetzky,[2] Houser[3] and others. The chemical and pharmacologic facts regarding this drug have been published in detail; one feels, therefore, that a brief record of personal experiences might be more valuable than a review of the voluminous literature, which has been very thoroughly covered recently by Schenck.[4]

Sulfanilamide has been of special value to us in the management of acute streptococcic infections of the sinuses and orbit; of the pharynx and its lymphatic drainage; of the larynx and trachea; and of the eustachian tube, middle ear and mastoid. So far we have not had occasion to use it for meningitis, although meningeal symptoms were allayed, in some of the conditions mentioned, during its use.

In most of these situations the temperature reaction was high, with a relatively slow rise in the white count, pointing to impaired resistance in the face of severe toxic infiltration. One is tempted to assume that streptococci slip off into the blood stream in many of these conditions, but are quickly picked up and destroyed in the lymph glands, liver and spleen. Resistance has had its initial blow in most instances from exposure and chilling of the body.

The nose, blocked by fiery red turbinates, readily stirs up severe acute maxillary, ethmoid or sphenoid infection; frontal invasion of this kind was rare in our group of cases. Some had been made worse by indiscriminate dousing with strong ephedrine solutions or proprietary oils containing menthol. It was of course necessary to follow the usual technique of shrinking to assist drainage, with steam

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*Read before the Western Section, American Laryngological, Rhinological and Otological Society, Spokane, January 29, 1939.

From the Department of Otolaryngology, University of Oregon Medical School.

inhalations or steam towels over the face; but mild, continuous dosage with sulfanilamide shortened the attack in most of these cases.

The scarlet throat, with neck movements made painful by swelling of deep cervical glands against the vertebral fascia, was helped by local hosing out with hot saline solution; but sulfanilamide in small doses, while not analgesic, nevertheless restored comfort and brought dangerous temperatures down within two or three days. Similar results were obtained in a number of rather violent cases of streptococcic tonsillitis, two with puzzling pseudomembranes.

Edema of the floor of the mouth, with upward swelling of the tongue, and with marked lingual tonsillitis, occurred once and seemed to be helped by sulfanilamide. Several times a somewhat alarming swelling over the arytenoids and false cords, accompanied by a very red trachea, yielded quickly to sulfanilamide therapy and steam inhalations.

Streptococcic invasion of the eustachian tube leads very swiftly to its closure and the rapid multiplication of these germs in the middle ear. All are familiar with this picture—the "red-hot" ear with bulged and scarlet drum, which fills so rapidly with toxic fluid that the tympanic membrane does not have time to soften and break as in milder infections, hence favoring the rapid spread of the infection to the mastoid cells, the sigmoid sinus, or even through the tegmen tympani to the dura. Twelve to twenty-four hours is ample time for these infections to spread through most of the air cells communicating with the middle ear. Sulfanilamide is not a substitute for myringotomy, which must be done early and repeated whenever necessary. But one finds this drug an invaluable aid in disease of the temporal bone due to streptococci and pneumococci. Fever comes down very quickly; the polymorphonuclear count drops down; and discharge and mastoid tenderness are rapidly diminished. We have found such results even in certain cases with positive blood culture pointing to invasion of the sigmoid. Other cases presenting complete radiographic clouding of all the mastoid cells, running torrents of thin red pus, have cleared up almost miraculously and operation has been avoided. We have had no personal experience with meningitis cases from this type of otitis since sulfanilamide has been available. It was helpful in shortening the duration of one case of erysipelas.

Thus summarizing the favorable results of many private and clinic cases, the question of dosage comes up. Here we are guided by the careful experimental studies of Osgood,[5] who tried various concentrations of sulfanilamide on cultures of living human blood

cells inoculated with streptococci. He finds that a concentration of 1/100,000 for streptococcus and 1/10,000 for pneumococcus is necessary within the bloodstream. In the concentrations, leucocytes will live and hemolysis will not occur. It is, however, necessary for this concentration to be maintained day and night, otherwise death of leucocytes and hemolysis of red cells will take place. Apparently there is no antiseptic or sterilizing action by the drug; it does not kill organisms, but seems either to neutralize or decrease the amount of their toxins, or at least to facilitate the production of anti-bodies called out by such toxins present in the blood and tissues.

Dosage to secure the concentration mentioned (for adults) is from five to ten grains by mouth, day and night, for the streptococcus. Ten times as much, with the addition of a small dose of type specific serum, is said to be needed for pneumococcus infections. We have not lately made any use of "prontosil"; Osgood recommends 0.8 per cent sulfanilamide in normal saline, subcutaneously, where vomiting precludes its administration by mouth. It should be continued as long as cultures are positive, unless contraindications to its administration are found. No other drugs, except for hypnotics or analgesics, should be given. Transfusions will be very helpful in severe cases. We have used them in some cases with positive bloodstream cultures.

Contraindications to the use of sulfanilamide may be gathered from the long list of complications ascribed to its excessive use, including agranulocytosis,[6] acute hemolytic anemia,[7, 8] cyanosis,[9] purpuric and scarlatiniform eruptions,[10] exfoliative dematitis,[11] toxic dermatosis,[12] peripheral neuritis,[13] photosensitization of the skin,[14] fever,[15] sulfhemoglobinemia,[16] optic neuritis,[17] and origination of psychoses.[18] This is a rather appalling list, and these things have been thoroughly reported. We have only observed a few difficulties, apparently due to individual sensitiveness to this agent: one case of brownish-red eruptions over the flexor surfaces of the arms and legs; two reporting slight breathlessness (hyperpnea)—both middle-aged people with slightly increased systolic pressure; and perhaps half a dozen in whom the white count dropped rather rapidly, with a loss of several hundred thousand red cells. These symptoms all disappeared within a few days after discontinuance of the drug, which in all our cases was given in limited though constant amounts, day and night.

We have found it desirable during sulfanilamide therapy to check the blood findings at least every forty-eight hours. A drop in the red count, especially if there is a sudden diminution in the total

number of white cells, is a danger signal that may point to the need for transfusion to stave off a hemolytic anemia.

Too many clinicians have been prone to rely on the drug alone, without, especially in acute otitis, resorting to surgery early enough. Surgery in otitis media means early and successful myringotomy, and in the neck may mean drainage of deep-seated infection. Sulfanilamide has the disagreeable habit of covering up mastoid symptoms, and when myringotomy has been too long deferred streptococcic invasion of the mastoid cells may have occurred already. In such cases, the osteomyelitic process may be masked by the drug, until sudden and severe temperature, chills, and high white count recurring, point to the onset of complications. Radiographs taken in such otitis cases, apparently convalescent, where the tympanic membrane has whitened and discharge has ceased, will occasionally show extensive destruction in the mastoid.

Nevertheless, a sufficient number of cases have been saved from operation—mastoidectomy, jugular ligation, orbital or pharyngeal abscess—to cause us to be very thankful for this powerful addition to our list of drugs.

Paul Leech, secretary of the Council of Pharmacy and Chemistry of the American Medical Association, stated recently that no other drug had been so rapidly and universally adopted and used in many years, and that in view of the careless and somewhat empirical fashion in which it has been employed by many, the number of complications due to its use has been surprisingly small.[19] One can scarcely agree with a dental surgeon who was recently reported in the press as stating that practically all maxillary suppuration after extraction of the upper molars has been prevented by the routine prophylactic administration of sulfanilamide. Its indiscriminate use by physicians or dentists unaware of its dangers, or by the public, requires rigid control.

Having avoided hitherto all details of specific cases in favor of the enunciation of principles derived from a fairly large series, may I be permitted to mention certain details of one very complex case? A woman past 50, with high blood pressure and marked cardiac incompensation, suffered a fracture of the orbit involving the ethmoid cells and antral roof, due to syncope with impact on the malar bone. After the initial nosebleed, no radiographs were taken. Increasing pain with temperature to 102 degrees after ten days followed her efforts to irrigate the nose with hot saline douches, at which time we were called in. The antrum and ethmoid were dark,

and chemosis, proptosis and limited movement of the eye were noted, which rapidly got worse, with a white count of 16,000 and temperature to 104 degrees. Sulfanilamide was administered, five grains by mouth every four hours day and night with daily white and red counts. This was continued for one week, with almost immediate cessation of pain, and return of temperature to normal in four days. Normal eye movements were gradually restored after six days, although after two months there is still sufficient latent hyperphoria to require the addition of prisms to the patient's correction. Here was a traumatic hemorrhage into the ethmoid, orbit and antrum, infected by the patient and causing orbital cellulitis and general symptoms so violent as to suggest the possibility of cavernous sinus invasion. The situation yielded rapidly to sulfanilamide, which was discontinued the moment a fall in the red count was noted. In view of the serious impairment of this patient's resistance, due to her previous serious cardiovascular condition, surgical attack on this type of sepsis would have been practically impossible.

Such life-saving instances might be multiplied; but they must not lead us to neglect of surgery or to the excessive or incautious use of this valuable agent.

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III

SULFANILAMIDE IN OTOLARYNGOLOGY*

JOHN G. McLAURIN, M.D.

DALLAS

It was early in 1935 that Gerhard Domagk of Germany reported a very startling chemotherapeutic success. He had injected hemolytic streptococci of human origin into the peritoneum of twenty-six mice and an hour and a half later twelve of them received by a stomach-tube a single dose of a red dye, that he afterward called prontosil. The drug had been synthetized by Meitzsch and Klarer, chemists quite familar with the various analine dyes. All of these twelve mice so treated survived—at any rate none died under seven days. Thirteen of the remaining fourteen mice that had not received the drug were dead within three days and the fourteenth mouse died on the fourth day. Thus he proved that the drug had an elective action on streptococcal infections. Later he demonstrated that it had some favorable effect in combating staphylococcal infections in the rabbit. At about the same time Levaditi and Vaisman in France used a similar compound synthetized by Gerard and obtained some curative results in mice, but their experiments were not so convincing. The bacteriacidal properties of some of the azo dyes had already been reported by Eisenberg as early as 1913, and the therapeutic possibilities were discussed by him. It had been shown by Horlein a quarter of a century ago that dyes have a sulphonamide radical had a special affinity for certains proteins. Prontosil had really been produced by Meitzsch and Klarer in 1932, but it was not until three years later that Domagk proved its astonishing protective action against streptococcal infection in mice and at that time gave the name of prontosil to the chemical agent. Levaditi and Vaisman, however, were the investigators that showed that it was a metabolic product of prontosil in the body that was responsible for the chemotherapeutic action. Colebrook and Kenny reported that the hydrochloride and the disodium salt of the drug possessed no bacteriostatic effects in vitro, but that the serums of patients treated with these dyes possessed bacteriostatic qualities against hemolytic streptococci. Perrin Long and Eleanor Bliss in their preliminary communications

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thought that they had noted bacteriostatic effects of sulfanilamide in vitro and suggested that the therapeutic dye compounds might be activated in vivo by reduction. Colebrook and his associates then reported that on the basis of further in vitro tests using whole blood and serum that the blood and serum of patients who had received the drug showed bactericidal as well as bacteriostatic qualities. S. M. Rosenthal then reported that sulfanilamide was bacteriacidal to pneumococci. Much work has been done by many other investigators in an effort to explain the satisfactory results obtained by the drug, but the mode of action of sulfanilamide is not yet positively known. Recently Bliss and Long have expressed the opinion that sulfanilamide inhibits the growth of certain bacteria in vitro and that in vivo it produces a change in the micro-organisms which permit them to be phagocytized by the white blood cells. These two men showed that the drug was bacteriostatic in concentrations of 1 to 10,000 in vitro and, therefore, tried to obtain a high concentration in blood of patients treated with the substance. They knew little about the absorption of the drug and practically nothing of the excretion of it, however, they found that some patients would tolerate a gram, 15 grains, to each 9 Kg. or 20 pounds of body weight for at least one month. My own experience is that very few patients can stand half that amount for even ten days. According to their statement some patients weighing 160 pounds could tolerate doses of 120 grains per day for thirty days. I have never tried to place patients on such large doses over a long period of time and personally feel that it would produce untoward symptoms in most patients and that such doses are really unjustified in view of satisfactory results obtained by much smaller doses. Some work done in the Department of Bacteriology, Hygiene, and Preventive Medicine at Baylor University under the direction of Dr. Hardy A. Kemp has failed to demonstrate antihemolytic, antifibrinolytic, or antitoxic effect of the drug. As a matter of fact no one knows at present just how the drug acts, but its value in the treatment of certain infections cannot be questioned. Some day its mode of action will probably be understood, but even if such should not be true, its value would not necessarily be doubted. Just who knows how quinin acts on the malarium plasmodium, but who would question its value in the treatment of malaria?

Practically all the work done at first with sulfanilamide was directed to the control of streptococcal infections especially those due to the Beta hemolytic strain, just as salvarsan was originally used only in the treatment of syphilis. Later salvarsan and neosalvarsan

were found beneficial in the treatment of some types of staphylococcal infections especially those of the urogenital tract, Vincent's angina, malaria and several other diseases. Very shortly it was realized that sulfanilamide had some satisfactory effect on controlling certain staphylococcal infections, and now it is being used with fairly satisfactory results in infections due to the gonococcus, meningococcus, Escherichia Coli, Clostridium Welchii, and certain strains of pneumococci, especially Type III. Just recently it has been reported that undulant fever can be satisfactorily treated by sulfanilamide. Many cases of trachoma have also yielded satisfactorily to this drug.

Harold R. Bohlman has recently reported the effective use of sulfanilamide in the control of gas gangrene, and concludes that the drug has a specific effect on gas bacilli, but the results may in part be due to checking symbiotic growth with the streptococcus.

Most of the early work was done with prontosil administered by needle. Prontosil was the trade name used by a certain chemical company for their product of a ruby red solution of sulphonamide (chemical formula of 4—sulphamido-phenyl—2 azo—7—acetylamino—1 hydoxy naphthalene 3, 6-disulphonic acid). It is employed in the form of a disodium salt in 2.5 per cent aqueous solution and can be given intravenously, intramuscularly, and even intrathecally. This form of the drug is valuable where a desperate infection exists and an immediate effect is required, or in cases where the patient cannot take drugs by mouth, however following the administration of the drug intravenously and sometimes intramuscularly there may be rather disagreeable reactions. Under ordinary conditions a somewhat altered form of the drug is now used. Its chemical formula is para-amino benzene sulphonamide and has been given the official name of *sulfanilamide*. It appears as a white crystalline powder and is usually manufactured in 5-grain tablets to be taken by mouth. It is insoluble in water, but soluble in mineral acids and caustic alkalis.

The otolaryngologist will find that he can use sulfanilamide to advantage in treating patients suffering with the following conditions: (1) Pharyngitis, especially of the ulcerative streptococcal type; (2) sinusitis with purulent or sero-purulent discharge; (3) rhinitis associated with a pharyngitis or sinusitis; (4) otitis media complicating a nasopharyngitis; (5) mastoiditis and cellulitis about mastoid wounds, especially when due to beta-hemolytic streptococci; (6) purulent conjunctivitis associated with upper respiratory infec-

tions; (7) laryngitis or tracheo-bronchitis associated with an active pharyngitis; (8) Ludwig's angina; (9) furunculosis of the outer ear canal; (10) acute cellulitis with or without furuncle formation of the upper lip, columella, wings of the nose or floor of the nose, and (11) cavernous sinus thrombosis. Complications such as pneumonia of certain types and meningitis of otogenic origin or the result of sinus infections, may also respond to the use of the drug. Pneumonia, Type III, has responded exceptionally well to sulfanilamide. A sufficient number of cases of pneumococcal meningitis have recovered under the use of the drug to make it always advisable to administer sulfanilamide to these cases. More instances of recovery of streptococcal meningitis have been reported where the drug has been used in the past three years, than had been reported for thirty years prior to the time the drug was available. A number of men have reported a high percentage of cures of meningococcic meningitis where sulfanilamide was used, and most of them have concluded that in these cases the oral administration of the drug is as valuable as when given parenterally. Two cases of brain abscess, due respectively to Type III pneumococcus and non-hemolytic streptococcus, were reported by Basman and Perley as having recovered by sulfanilamide therapy. Dr. Paul C. Bucy has just reported the recovery of a hemolytic streptococcal brain abscess by aspiration of the abscess combined with sulfanilamide administered parenterally and orally.

During the past year we have treated 180 cases of acute ulcerative streptococcal pharyngitis with sulfanilamide and we feel that the response to the drug has been most satisfactory. Cultures showed the predominating organism to be a short-chain streptococcus, more often non-hemolytic but sometimes hemolytic. In many instances the speed of recovery has been rather startling, the patient being free of fever and well on to recovery in from twenty-four to forty-eight hours. In practically all cases we have felt that the use of the drug was responsible for materially reducing the duration of the disease and that it prevented many complications that otherwise would probably have occurred. We have been most gratified by the results we have gotten when the drug was judiciously used. Our experience has been that it was far better to have the patients hospitalized when possible, because in that way daily blood counts could be taken and the sulfanilamide coefficient of the blood could be determined as often as advisable. In addition to these observations, the patient could be kept absolutely quiet, thereby minimizing many of the disagreeable reactions of the drug. Of course, the regular charting of the temperature, pulse and respiration was very worth while. The

necessary dietary regime could also be followed better in a hospital. Many patients, however, do not go to the hospital while taking the drug, but in such cases it is better to take a complete blood count before starting the drug and then every second day while the drug is being given in fairly large doses. In an adult case we usually start the dosage at twenty grains every four hours during the day time for three or four doses and then drop to a dose of ten grains every four hours while awake. Most acute cases are under control within forty-eight hours from the time this regime is started. We do not feel that it is advisable to awaken our patients during the night to give the drug and as a rule give them some pheno-barbital preparation if necessary to make them sleep, however the drug itself seems to make many of them sleep a great part of the time. Some rather persistent cases of purulent otitis media complicating these cases of pharyngitis have cleared up and stopped draining soon after the drug was started. Acute catarrhal conjuctivitis complicating cases of acute rhinitis have responded quite favorably to the drug. One very severe case of cellulitis involving the upper lip and external nose cleared up under the use of the drug. We feel that this case would not have cleared without the sulfanilamide. Another very extensive streptococcal infection of the outer ear canal and face cleared up rather quickly after the drug was used. In this case there was never any purulent drainage. Cultures from a serous drainage showed a short chain streptococcus.

In cases that show a tendency to have recurrences we keep the patients on small doses of the drug for a long period of time. Five grains every four hours or twenty grains a day will usually be sufficient to clear up these cases eventually and prevent recurrences of their troubles. Chronic cases should be given small doses of the drug over a long period of time unless there develops some contra-indications to its use. A few more things should be said about dosage. Some men believe in giving exceedingly large doses—as much as 250 grains of sulfanilamide in one dose, while others are apparently getting good results with rather small doses. Marcel Pinard, of Paris, feels that it is never necessary to give more than forty-five grains a day and he reports excellent results in handling acute cases with such doses. I would like to quote from a letter that I received from Dr. Perrin H. Long of Johns Hopkins Hospital, as follows: "At the present time, in patients acutely ill with hemolytic streptococcal infections and who are in bed, we advise the following dosage: In adults twelve to sixteen tablets for the initial dose, followed by three tablets every four hours until a definite clinical effect is

obtained, at which time the dosage is cut down. In children weighing from fifty to ninety pounds the initial dose is six to ten tablets followed by two every four hours. Children weighing from twenty-five to fifty pounds get four to six tablets for the first dose, followed by one or two tablets every four hours. These doses are too large for ambulatory patients, because if one is up and about, one notices a disturbing sensation of dizziness and definite nausea following these larger doses. However, for rapid clinical effects in acutely ill patients, these doses are extremely efficient. The reason why we start with large initial doses is that we have found in conjunction with Dr. E. K. Marshall, that a blood sulfanilamide level of 10 to 15 mgm. per 100 cc. represents an effective concentration of the drug. The method for determining the concentration was devised by Dr. Marshall.

"The dosage which I have outlined usually results in mild toxic symptoms, slight nausea and dizziness are common, and varying degrees of cyanosis which result from the formation of methemoglobinemia is also common, but we disregard it entirely. Every patient receiving sulfanilamide has a fall in their CO_2 combining power, and in four or five instances clinical acidosis has resulted. For this reason in adults we generally give ten grains of soda bicarbonate with each dose of sulfanilamide, and in children five grains. Occasionally fever results from the prolonged use of the drug, and when you think that has occurred, the best thing to do is to stop the drug, and the temperature will come down to normal within twenty-four hours. It is important to space it at four hourly intervals because absorption is complete at that time, and excretion is at its height. The chemical is excreted mainly by the kidneys. We have noted that the damaged kidney that shows decreased function or nitrogen retention does not excrete the drug readily. Therefore, in those individuals who have impaired renal function, we quantitate the blood sulfanilamide level daily in order to prevent it from piling up in the blood, and when we reach a 15 mgm. level, we stop and wait until the level falls to 8 or 10 mgm. before starting the drug again."

Dr. Long outlined the dosage for children and I want to bring out the point that in our own experience children, suffering from the acute infections mentioned, apparently respond even more promptly to sulfanilamide therapy than do adults. Neither do they seem to be so definitely disturbed by the toxic effects of the drug. When treating patients at home we usually give them written instructions so that they can make no mistake in the plan of therapy intended.

I have been able to touch on only the high points of this subject, but the many uses of the drug and its mode of administration have been discussed by hundreds of essayists. Nearly every doctor has arrived at some definite conclusions himself as to how he shall use sulfanilamide. In discussing this drug it is important to call attention to some of the toxic symptoms produced. These can de divided into two general classifications: First, those that are apparently due to the inherent properties of the compound, varying in proportion to the dosage and according to the individual tolerance, and; second, those due to some sensitivity phenomenon or idiosyncrasy manifested by skin reactions or changes in the blood picture with a temporary damage to the hemopoetic system. Under the first heading would be listed headache, nervousness, mental confusion, nausea, anorexia, vertigo, weakness, lassitude, precordial pain, abdominal discomfort and aching of the extremities. These toxic symptoms are common and are usually not severe enough to warrant discontinuance of the drug. Probably ten per cent of these patients will have one or more of these symptoms so aggravated that it will be necessary to discontinue treatment. I have seen one patient who developed such a severe headache after taking twenty grains of the drug that it was necessary to give large doses of opiates and discontinue the drug. Another patient suffered intensely with headache and became so confused that she wandered about the halls of the hospital and did not know later that she had been out of bed. All toxic symptoms disappeared within forty-eight hours after the drug was discontinued. In all probability, excluding individual susceptibilities, the toxic symptoms vary in degree in proportion to the blood level of sulfanilamide. Undernourished patients, those suffering from malignancies, and patients with poor renal function do not tolerate the drug very well. One of the most common manifestations of sulfanilamide therapy is cyanosis. It has been ascribed to the formation in the blood of sulfhemoglobin and in some instances to methemoglobin. Sulfhemoglobin may occasionally be demonstrated in the blood of patients who have been taking sulphates, but in all probability it does not appear in the blood nearly so often as is generally supposed. Some investigators have only rarely demonstrated sulfhemoglobin in patients taking the drug. Sulfhemoglobin is more likely to develop in those cases where laxatives are given with resulting liquid stools and considerable gas formation in the colon. In such instances the normal absorption of protein products in the small intestine is diminished, and this causes an increased putrefaction in the large bowel with the formation of excessive amounts of hydrogen sulfide. The hemoglobin combines with the hydrogen sulfide ab-

sorbed from the bowel to form sulfhemoglobin. The drug may serve as a catalyzing agent, but sulfhemoglobin has been demonstrated in cases suffering with colitis long before sulfanilamide was used.

Marshall and Walzl investigated the oxygen capacity and total iron pigment in patients receiving sulfanilamide. They reported that the cyanosis was probably not due to non-functioning iron pigment such as sulfhemoglobin or methemoglobin and indicated that the explanation of cyanosis was yet undetermined, but that probably it was the result of a black oxydation product of the drug that directly stained the red blood cells. It is probable that there is also some staining of the intima of the vessels. Wendall could not even produce sulfhemoglobin in animals where large doses of the drug were given. Methemoglobin has been more definitely demonstrated by investigators generally and as a result of that fact Williams and Challis suggested the administration of methylene blue to combat the formation of methemoglobin. They showed that a single injection of 1 mg. of methylene blue per kilogram of body weight reduced a methemoglobin of twenty per cent to one of three per cent within forty-five minutes from the time of injection. It would appear that by this plan of controlling the methemoglobin it would be possible to increase the sulfanilamide concentration in the blood and thereby make the sulfanilamide therapy more effective. This is still a mooted question and requires further investigation. My experience has been that in acute infections treated by sulfanilamide the most prompt responses have appeared in those cases where some cyanosis developed.

It is estimated that six per cent of all cases treated with the drug show some toxic skin lesions. Various types of skin eruptions have resulted from the use of the drug, the most frequent one being a morbilliform rash, but the lesions may be papular and may or may not itch. In some instances the skin lesions are distinctly hemorrhagic or purpuric. Those portions of the skin exposed to the sun-light seem to be influenced more readily because of a photo-sensitizing agent produced by the drug. In some instances urticarial reactions have occurred, which to us indicated an allergic reaction to the drug. I saw in consultation one case of exfoliative dermatitis associated with marked hemolytic anemia. The red cell count dropped down to about 900,000. This patient was being treated for gonorrhea with sulfanilamide, and for syphilis with neoarsphenamin at the same time. Of course, both drugs were discontinued, and after several transfusions the patient made a nice recovery. Southworth reported fifteen cases of acidosis. There is

no doubt that in some patients receiving the drug the CO_2 combining power is markedly reduced. The mechanism of the acidosis is apparently due to the excretion of base, especially sodium and potassium ions. The oral administration of sodium bicarbonate or some similar alkalanizing agent is therefore advisable. Fever has also been noted after the administration of sulfanilamide and it is sometimes quite difficult to know whether the fever is the result of the drug or of the disease being treated. Fever due to the drug usually subsides in from twenty-four to forty-eight hours after discontinuance of the drug, however, if certain skin lesions mentioned above are present, the fever may continue for a much longer period.

I wish to report some observations on the erythrocyte sedimentation rate in cases where sulfanilamide was given. The rate was estimated before the drug was started and again when cyanosis was first evident. The rate was invariably markedly increased—sometimes with a rate of 50 to 60 mm. in the first hour. It is possible that the infection was responsible for the increased rate, but cases of the same infection, proven by culture and clinical observation, treated without sulfanilamide, showed very little increase in the rate—never more than 25 mm. in the first hour. The rate was taken in these cases when treatment was started and again four or five days later, the usual time in which the cyanosis would have developed if the cases had received sulfanilamide.

All of the toxic symptoms that I have discussed are themselves disagreeable and may in some instances necessitate the discontinuance of the drug. Certain infrequent alterations in the blood picture demand an immediate withdrawal of the drug. Hemolytic anemia must always be kept in mind and if a rapid drop in the red cell count is occurring, the drug must be discontinued. It is estimated that in only two cases out of a thousand receiving the drug will transfusions become necessary to combat the rapid loss in the red cell count. In extremely rare instances agranulocytosis may occur necessitating prompt discontinuance of the drug and the use of transfusions.

In conclusion, I wish to call attention to the fact that recently there has appeared a new drug on the market known as neo-prontosil. It appears on the market in five-grain tablets or capsules—the latter having the advantage of not staining the mouth and fingers. The drug is quickly absorbed in the gastro-intestinal tract and is eliminated very rapidly through the kidneys. It can be detected in the urine within four hours of its ingestion. It is sup-

posed to be seven times less toxic than sulfanilamide, but just as effective as that drug. Where it is used, however, the free sulfanilamide in the blood has never been reported higher than 3.6 mg. per 100 cc., though dosages as high as 6 gm. per day have been given. The most recent related product to sulfanilamide is sulfapyridine, which is supposed to be more effective in controlling certain types of pneumonia than sulfanilamide itself. The Council of Pharmacy and Chemistry has given the product its official name as above, but up to this time the drug is not on the market and the government has not licensed it for interstate sale. I do think, however, that we have just started to realize the importance of chemotherapy and that within a reasonable time there will probably be a new drug, likely related to sulfanilamide, that will produce all of its beneficial effects without resultant disagreeable toxic symptoms.

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IV

THE AIR CELLS IN THE PETROUS TEMPORAL BONE IN A CHILD OF THIRTEEN YEARS*

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This is an anatomical study of air cells of the petrous temporal bone in a child of thirteen years and of their relation to the middle ear. It is the second part of an investigation of the tubal cells as observed at various ages. Permit me one or two introductory remarks regarding the petrous temporal as a whole, which bone I regard as the most interesting in the skull—because of its part in the formation of the cranium and of its contents.

The petrous bone juts out at an angle at the base of the skull. Its apex (apex pyramidis) is inserted in the angle between the basi-occipital and sphenoid, leaving an irregular area filled with fibro-cartilage (foramen lacerum). It alters in size, form and position to conform to the growth of the cranium. From measurements I have found its length in the adult, on the average, one and one-half times as great as that of the bone in the infant, and its width virtually doubled. Both progress up to and even beyond puberty to a still undetermined age. But it is not the entire petrous bone which enlarges. The membranous labyrinth and the bony capsule which encloses it does not materially increase in size after birth. Thus, while the labyrinth and its immediate surroundings remain unaltered after birth, a part related to the growth of the skull enlarges due to cartilaginous and bone increase at its apex and to bone deposited under the periosteum. The air cells and bony trabeculae participate in this enlargement.

Tubal cells have long been recognized, but they have received little attention from anatomists, and it is only a little more than a decade ago that otologists became impressed with the relation of these cells to chronic ear discharge and to meningitis.

It is well known that there are differences in the extent, size and location of these cells in different individuals. This specific case was chosen from a large series, since it presented well marked air cells

*Presented before the Meeting of the Chicago Laryngological and Otological Society, December 5, 1938.

and other anatomical features which we believe to be typical. It is probable that ultimately separate types of petrous bone will be recognized as has come about with regard to the mastoid. But we believe that the general pattern to be described will hold good for all.

Though the series were derived from postmortem cases, we are not now concerned with the diseases that may here occur nor with the question whether such infections most frequently arise as a direct invasion from the middle ear or through vascular channels. It is sufficient to say that the examination shows that the infection may come not only from the general vascular system but may spread from the middle ear. In subacute cases the pathologic reaction is often marked adjoining vascular areas connecting with or adjacent to an infected tympanic space or infected areas near the carotid canal or eustachian tube, without noticeable bone destruction under the membrane of the middle ear.

In a study of the air cells in a child it was necessary to consider how the cells are formed. To discuss this lies outside the limits of this demonstration. However, it may be said while our observations point in some cases to a resorption of the bone by osteoclasts, the absence of osteoclasts in many specimens suggests that a prior change takes place in the adjacent bone, a lysis, it may be halisteresis, resulting in easier penetration by blood vessels with accompanying connective tissue and epithelium, into the adjacent marrow spaces. Ultimately the connective tissue is absorbed, leaving a thing layer of epithelium covering the bone. In short, a development similar to that seen in the mastoid. Beyond these observations lies the as yet unanswerable factor of cell development and how far it can be influenced by environmental circumstances, such as past or present pathological conditions existing in the middle ear.

The description of tubal cells given by Bezold and by Siebenmann towards the end of last century, based chiefly on corrosion preparations, are still copied iin our textbooks of anatomy and otology. They demonstrated the shape and grouping of these cells and of certain small pneumatic spaces situated outside in the mastoid region. Within recent years numerous reports have been made on the air cells of the petrous pyramid, their size and location. These reports are chiefly based on dissection, corrosion preparations and roentgenography, all of which have inherent in them the obvious defects of the method used. Any method allowing only gross examination is entirely inadequate; the intricacy and the minuteness of numerous communications between spaces and the delicacy of the trabeculae which separate air cells from marrow spaces render impos-

sible the absolute identification of the two kinds of spaces. The corrosion method is undependable, since the material employed may spread only to the air cells immediately adjacent to the larger cavities. The dependability of roentgenograms for anatomic study of the air cells is greatly reduced by the deep and protected position of the petrous portion of the temporal bone and the absence of any constant pattern of pneumatization. The microscopic examination of serial sections of temporal bones is the only method which permits accurate identification of pneumatic and diploetic spaces in the bone. The serious shortcomings of this method is the difficulty of visualizing in three dimensions the form and the relations of the cavities and of their communications seen microscopically in only two dimensions and in a single section at a time. The method we have employed is that of reconstruction by the Born wax-plate method, which is recognized as possessing many advantages over the methods mentioned above. Thus, it enables us definitely to differentiate by their histological characters the air cells from marrow cells, to establish clearly their anatomical location and by suitable enlargements of microscopic sections so to reconstruct the part as to obtain a representation of the cells in a three dimensional form.

The location of the air cells and their relation to the marrow cells is of importance. In the specimen we are illustrating, the marrow cells predominate in the superior part of the pyramid. In sectioning from above down in the horizontal plane the first air cell comes into view about three and one-half or four mm. below the superior petrosal sinus. The marrow cells diminish in number as we proceed downwards, until in the inferior area the cells are predominantly air cells. As clinicians we are too apt to forget that marrow cells in this region as elsewhere have a defensive function equipped to deal with an infection reaching this area. In specimens in which evidence of otitis media or of basal meningitis was seen, our sections show that the marrow tissue is greatly increased, so great in some cases as to conceal completely the marrow spaces with their dilated capillaries.

The degree of pneumatization of the petrous pyramid varies greatly in different subjects. The air cells may come from two sources: (1) extension from mastoid cells spreading over the semicircular canals (epitympanic cells); (2) cells arising, independently of the mastoid cells, called tubal cells from their proximity to the upper end of the eustachian tube. These tubal cells may be widespread, but they lie chiefly in the inferior part of the pyramid and are in close proximity to the carotid canal. In the microscopic study

of serial sections, the relationships of the air cells appear hopelessly confusing. But when viewed in the reconstruction they are found to assume a fundamentally simple pattern, related chiefly to the carotid canal, and to be outgrowths from the postero-medial wall of the tympanic cavity near the orifice of the auditory tube. This relationship of the tubal cells to the carotid canal was noted by Bezold and Siebenmann, and has been frequently referred to by other writers.

The epitympanic cells are outgrowths from mastoid cells and can be traced there. In our specimen there is no communication between the epitympanic extensions from the mastoid cells and the air cells derived from the tubal cells; the solid bone of the otic capsule seems to provide an effective barrier against such an intercommunication. The cells of the epitympanic mastoid group penetrate the bone of the canalicular area, and in this specimen almost surround the superior semicircular canal. They lie on both sides of the subarcuate fossa.

No anastomosis occurs between any air spaces in the apex and similar spaces in the sphenoid and occipital bones.

There are air cells in close relation to the auditory tube, but only by position, not by origin; these extend along the tube from their origin in the hypotympanic region. We found no cells arise directly from the tube itself.

When is pneumatization complete in the petrous temporal? To this no answer can at present be given. We may look for some guidance in the mastoid area where it is agreed that the general pattern of pneumatization is practically complete at the age of five, and thereafter only a slow increase in their size. In the few cases we have specially examined there was a correlation between well developed tubal cells and well developed mastoid cells. How general this correlation is, we have no evidence.

SUMMARY

1. Air cells in the petrous were well developed in this 13-year-old child.

2. They come from two independent sources: (1) As extension of the mastoid cells, (2) tubal cells from the hypotympanic space of the middle ear. They do not communicate with each other.

3. The tubal cells lie chiefly in the inferior part of the petrous arc and are related to the carotid canal. They open into the hypotympanic space by a distinct channel or opening.

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V

NICOTINIC ACID AND THE EIGHTH NERVE* A PRELIMINARY REPORT†

GRANT SELFRIDGE, M.D.

SAN FRANCISCO

Nicotinic acid, one of the factors of the vitamin B_2 complex, has very recently been brought to the attention of the medical profession and the laity by various writers as the substance long sought for the cure of pellagra. This is now definitely recognized as a nutritional disease and affecting some four hundred thousand people in the Southern States with seven thousand deaths annually. The results following its use frequently appear in most dramatic fashion and yet further reports in medical literature suggest that other unknown factors come into the picture![1]

In February, 1937, the "filtrate factors" of the vitamin B complex were furnished me by the Vitab Company of Emeryville, California. This is that portion of their rice bran B complex from which B_1 (thiamin N. N. R.) and B_2 (riboflavin) had been removed with Fuller's earth. This fraction, although treated with Fuller's earth, was known to contain some B_6. It was also known to contain nicotinic acid. The material was used experimentally in several cases of chronic progressive deafness because the bone conduction curve showed a marked drop at 1024 and 2048 cycles. Its use was suggested by Covell's discovery of a marked and rapid degeneration of the eighth nerve in a series of vitamin B deficiency dermatitis chicks furnished by Jukes (Agricultural College, University of California). The use of this material apparently straightened up some portions of these dips shown in the bone conduction curves.

Covell's studies of the different fractions of the B complex, to be published shortly, indicate that lack of the filtrate factors (a deficiency of which leads to chick dermatitis, and probably also to black tongue in the dog) causes a greater amount of degeneration in both branches of the eighth nerve than is seen in the B_1 deficient rat. The

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*From the Harriman Research Laboratories, Southern Pacific General Hospital.

†A second study is in progress on the comparative value of B_1, nicotinic acid, and sodium nicotinate, based on urinary analysis for B_1 and porphyrin.

use of nicotinic acid in deaf humans after the age of 55 would seem to be justified rather than B_1.

In two series of chicks that had been on a recovery diet for several weeks, although they looked externally like normals, there appeared some continued destruction of the nerve when it was studied in the laboratory. There were, however, several areas in the nerve that gave evidence of a regeneration taking place. It seems evident, therefore, that further recovery would have followed had the birds been on the recovery diet for a period of several months rather than a few weeks. These dermatitis chicks have a very stormy short life and are desperately ill. Among other things, the posture indicates an involvement of the sciatic nerve, in fact, Covell has found involvement of all the cranial nerves, cord, etc., in many of the birds examined.

It must be understood at the outset that no claim is made that the filtrate factors are the only ones concerned in eighth nerve involvement. In fact, Covell has not only shown that this exists in animals deficient in the different portions of the B complex, but he has also found degeneration of the nerve in animals deficient in other vitamins. Mellanby[2] also calls particular attention to degeneration of the eighth nerve in vitamin A deficiency. At the recent Congress of Physiologists in Zurich (August, 1938), he reported finding hyperostosis of the modiolus with definite damage of the eighth nerve (pressure).

Our observation so far appears to show a more rapid improvement in the air conduction and bone conduction audiometric curves with nicotinic acid than with B_1 or riboflavin. In Case 1 several injections of B_1 (500 I. U.) did not produce any change, whereas the use of nicotinic acid was followed in ninety days by marked improvement. Perhaps the use of 3000 I. U. of B_1 parenterally might have occasioned a prompter change. At a later date I will report the results of such dosage.

I have endeavored to show in previous articles that degeneration of the eighth nerve is related to nutritional deficiencies, and even though the missing factor or factors are supplied, the dietary errors must be corrected! In discussing the diet in the treatment and prevention of pellagra, Sebrell[1] says "Permanent control can be obtained only by bringing about permanent changes in the dietary habits."

In this present study, nicotinamide and nicotinic acid have been the substances used. In general, these substances were given by mouth, although in a number of cases I have used the amide parenterally (ten

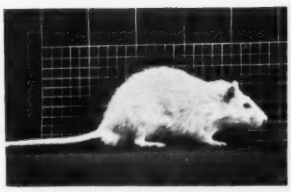

Fig. 1A. Normal rat.

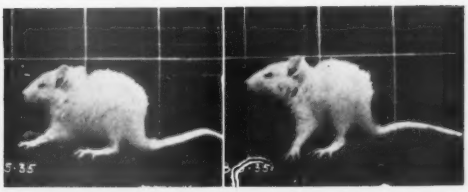

Fig. 1B. These two pictures represent a rat in acute B_1 deficiency. His posture indicates an impending attack of vertigo.

Fig. 2A. Jukes' dermatitis chick (filtrate factor). Note beak dermatitis.

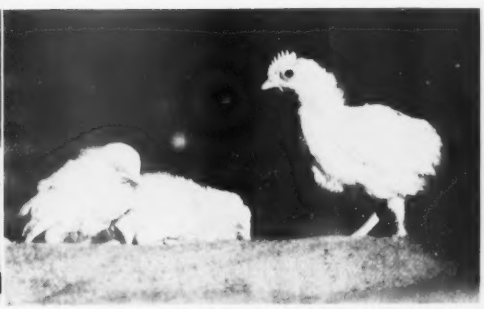

Fig. 2B. Jukes' dermatitis chicks and control.

or more intramuscular injections, 30 mgm. per cc.) before using it by mouth. Following the injections there appeared in some cases a bit quicker response than by mouth. It is therefore quite important in all cases of beginning loss of high tones to consider possible allergy, drugs, syphilis, arteriosclerois, water balance, hypothyroidism and wherever possible a thorough physical examination by a competent internist. A urine study for porphyrin may be helpful as it is frequently found in pellagra, and therefore a further indication for nicotinic acid. Likewise a urine check for B_1 to determine whether the individual has a normal amount is equally important in deciding which substance to use.

* * * *

Little or nothing is known about the physiology of nicotinic acid. Peters and his co-workers of Oxford University have reported tissue respiration studies on B_1 that have been confirmed by Elvehjem of Wisconsin. "They think B_1 functions as a co-enzyme in the metabolism of carbohydrate, more specifically in the exudative breakdown of pyruvic acid."[3] Peters[4] thinks this process explains, by the accumulation of lactic acid in certain areas of the brain, the opisthotonus seen in B_1 deficient pigeons. So far no tissue studies have been carried on in relation to nicotinic acid. When they are done, findings similar to Peters' but more exaggerated may be found. Apparently its lack in pellagrins is definitely related to disturbance in water balance and probably will explain the tremendous edema found in the gastric and duodenal mucosa observed by Lucia (University of California Medical School) in some of his autopsies on alcoholic pellagrins.

Vilter, Spies and Mathews[5] say, "Since there is no evidence that pyridine can be formed in the human body, it is evident that nicotinic acid or its amide must be an important article of human diet and that it is apparently necessary for the normal health of the mucosa of the mouth and intestine, nervous system, and skin, since these all are impaired in pellagra and improved by the ingestion of nicotinic acid or the amide. It must be necessary for the muscles also, and probably for the liver and some other organs, since it is an essential part of co-zymase and of the co-enzyme (Warburg et al.) of these tissues and organs."

The most striking reaction following the ingestion of nicotinic acid in doses as small as thirty milligrams daily, particularly in vasomotor unstables (and it occurs almost immediately), is a marked flushing of the skin (vasodilator) accompanied by a sense of heat. This is noticed particularly in the face, but occurs in other parts of the body. Recently, cases taking sodium nicotinate have shown in-

tense redness of the entire body, the reaction in several instances lasting three to five hours, particularly when taken after breakfast.

Fouts and his co-workers[6] called attention to the skin reaction in 1937, and later Dann and his co-workers also called attention to this. I observed this reaction before the appearance of the article by Spies and co-workers and following its use in cases of eighth nerve degeneration.

Spies, Bean, and Stone[7] report its effects on one hundred non-pellagric cases. They state "The oral administration of large quantities of nicotinic acid is followed by flushing, burning, itching, and an increased sensation of local heat in the skin. This finding apparently can be produced in all persons, although the amount required to produce it is extremely variable at different times in the same person and from one person to another." Further on they state "Large doses of nicotinic acid suggested a parasympathetic-like action, similar to that produced by the acetylcholine group of chemicals."

There appears little doubt that nicotinic acid acts as a vasodilator and I have accumulated some evidence that this vasodilator effect not only influences the eighth nerve, but appears also to play a part in removing calcium in the adhesions around the foot plate of the stapes in chronic progressive deafness and perhaps also in early cases of beginning bony fixation in true otosclerosis.

CONCERNING CASE REPORTS

During the past nine months (from December, 1937) some thirty cases have been treated with nicotinic acid, nicotinamide, and quite recently sodium nicotinate has been used. The ages of the cases range from twenty-one to seventy-seven years. In many the results are very striking; in all cases, where the audiogram shows a gradual decline of the tone scale, one is I believe justified in making use of these fractions because while the improvement in the audiometric curve may not be very marked, the physical and mental improvement is sufficient justification.

Indeed, any measures tending to ward off senescence are justifiable, and evidence is slowly accumulating that several components of the vitamin B complex play a definite part in the advancing of age.

CASE 1.—Mr. W. A., age 21; December 13, 1937.

Complaints—Earache in left ear: Patient said he did not hear well.

History—Deafness: An audiogram taken on December 20, 1937, showed a drop in the high tones of both ears, though more marked on the left side. An

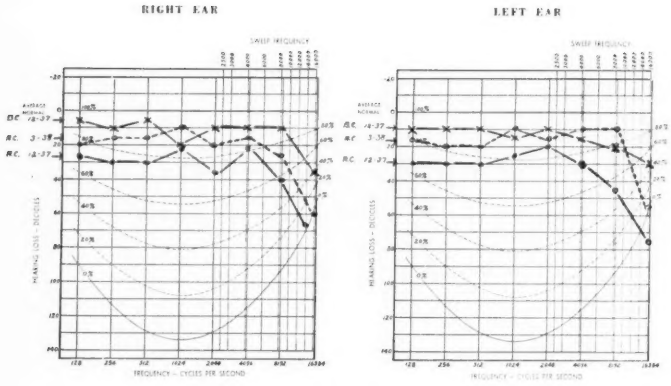

Fig. 3. Case 1

audiogram made of both ears three years before in 1934 showed nearly a normal curve. (See operations).

Diet: His dietary habits have been fair, although perhaps too much carbohydrates, little or no milk, and low in vitamins B and C.

Operations: Patient was operated on for acute right mastoiditis on December 26, 1923. Had a reinfection of the right mastoid on December 12, 1925; it was opened and drained. Tonsils removed in 1934. Had acute otitis (left) with incision of drum in 1935.

Diseases: German measles in 1936.

Examination—Physical: No physical examination has been done. Patient has been in excellent health all his life with an occasional lower respiratory involvement (bronchitis).

Ears: There was some secretion in the canal. Drum membrane looked more like a ruptured herpetic bleb. (The secretion lasted a couple of days and the normal luster returned in a day or two).

Tonsils: Out.

Treatment—December 20, 1937: Given six injections of B_1 solution, 500 I. U., one every other day. (No change in his audiogram).

January 1, 1938: Given nicotinic acid, 60 mgm., t.i.d (to May 13, 1938).

Comments: In spite of two involvements of the right mastoid, an audiogram made in 1934 showed his hearing curve of both ears (A. C.) to be about normal, and the upper limit of audibility of both ears was 17,000 cycles. It is hardly likely that the episodes in the left ear would explain the drop in the right ear.

The Sonotone Audiometer, which I now use, is calibrated for a sound-proof room and my testing room is not entirely 100 per cent sound proof. The "high

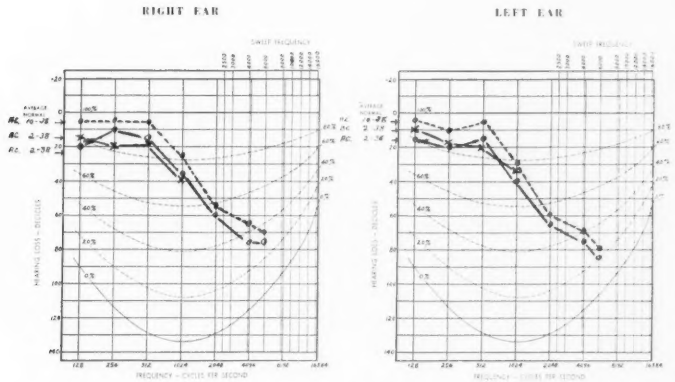

Fig. 4. Case 2

sweep" frequencies are not calibrated, and while the noise which penetrates my testing room is low frequency in character, it effects high frequencies adversely. Inasmuch as this audiometer is not designed to test hearing loss at high frequencies and as the high sweep is incorporated in the instrument only to estimate the upper limit of hearing, due regard should be had to apparent high tone loss readings, especially when the scale up to 8192 cycles appears normal.

It appears probable that the improvement shown in his last audiogram is definitely related to the use of nicotinic acid (one of the fractions of the B_2 complex).

Case 2.—Mr. H. J. E., age 39; November 2, 1937.

Complaints—Deafness: Both ears; diagnosed at the Mayo Clinic as nerve deafness.

History—Deafness: First noticed at the age of 23 years. Tinnitus was noticed first in 1936 (high-pitched in character). No vertigo or nausea. Does not hear better in noise. Is confused when several people are talking. Hears fairly well over the telephone and radio. (In the army at the front, he was a gunner, and served 75 mm. guns. The gun discharges were so noisy that he and his mates filled their ears with mud to lessen the concussion from the guns).

Allergy: None.

Diet: Poor diet all his life. Diet consisted mainly of potatoes, cabbage, salt pork, beans, few eggs, no fruits, milk, and whatever game his father was able to shoot. Later, when working as a printer's assistant, his diet was bread, meat and coffee. When overseas, the army food was principally white bread, beef, coffee; no beans, butter or eggs.

Diseases: Childhood diseases were scarlet fever, measles, mumps, whooping cough, and tonsillitis, followed by acute nephritis at the age of 8 years. Had an inflamed caecum, for which he spent one month in a hospital. Mild influenza at 18 years.

Examination—Physical: General health is good. No colds. Is chronically constipated.

Weight: 175 pounds.

Height: Five feet 10½ inches.

Ears: Drums very opaque, retracted, and light spot entirely absent.

Tubes: Open.

Nose: No abnormality. No discharge anteriorly or posteriorly.

Laboratory Data—Blood Count: Hgb. 100 per cent.

Treatment—November 2, 1937: Given injections of riboflavin "Roche" five times a week. (A 0.05 per cent sterile aqueous solution) (to January 4, 1938).

January 4, 1938: Given nicotinic acid, 60 mgm. daily.

April 4, 1938: Nicotinic acid increased to 60 mgm., night and morning (to August 16, 1938).

August 16, 1938: Given nicotinamide, 60 mgm., t.i.d. (to October 24, 1938).

October 24, 1938: Given sodium nicotinate, 120 mgm., night and morning (to date).

Comments: The result of the injections of riboflavin was that it apparently made no change in the audiogram, but caused improvement in the patient's feeling of well-being.

The use of the various preparations—nicotinic acid, nicotinamide, and sodium nicotinate*—appears to have produced a steady improvement in the hearing. Whether further improvement will continue remains to be seen.

CASE 3.—Mrs. H. B., age 43; October 12, 1937.

Complaints—Deafness: Both ears.

History—Deafness: First noticed difficulty in hearing three years ago. Deafness apparently not associated with colds, though she had earache as a child. Never had ear abscesses. Patient has no tinnitus.

Allergy: No hay fever or asthma.

Diet: Definitely low in vitamins B and C, and possibly A. Milk and eggs several times weekly, cheese seldom. Leafy salads and citrus fruit daily at present.

Catamenia: Normal; no evidence of menopause.

Operations: None.

Examination—Physical: General health good; only occasional fatigue. No constipation. No tobacco, alcohol, or drugs.

Weight: One hundred twenty-one pounds.

Height: Five feet three inches.

Ears: Slight retraction; broken light spot.

Tubes: Open.

Nose: Slight anterior deviation of septum on left side. Pharyngoscope examination shows posterior ends of both middle turbinates to be whitish; edema.

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*These preparations, through the courtesy of Merck and Company, Inc., have made this study possible.

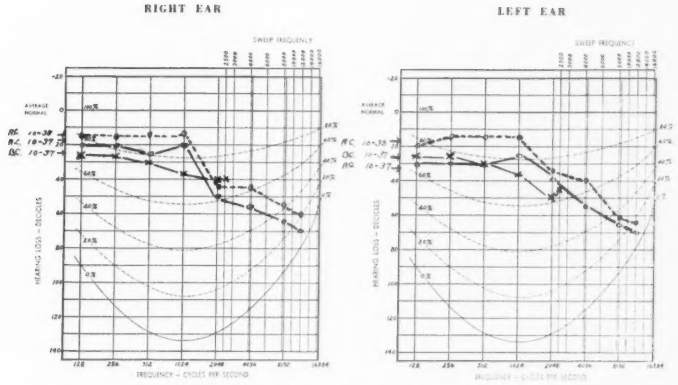

Fig. 5. Case 3

Tonsils: Still present; no infection.

Laboratory Data—B. M. R.: Minus 5 per cent.

Blood Count: Hgb., 61 per cent RBC, 4,000,000; WBC, 6,700 (October 20, 1937). Hgb., 84 per cent; RBC, 4,290,000; WBC, 7,800 (October 31, 1938).

Treatment—October 25, 1937: Given reduced iron, 0.6 gm., t.i.d., and injections of liver extract at seven to ten day intervals (to March 8, 1938).

March 8, 1938: Liver injections and iron discontinued and Lextron capsules substituted (to May 10, 1938).

May 10, 1938: Given nicotinic acid, 60 mgm., t.i.d. (to October 31, 1938).

October 31, 1938: To continue with nicotinic acid, same dosage.

Comments: The change in this patient's blood picture and physical well-being is quite obvious, and speaks improvement.

CASE 4.—Mr. N. F., age 56; April 15, 1937.

Complaints—Deafness: Both ears.

History—Deafness: Noticed deafness five years ago. No history of any involvement of the middle ear. No deafness in the family, except father at the age of 85. Patient has no tinnitus. Patient is a civil engineer by profession with the Southern Pacific Railroad Company, exposed to considerable noise.

Allergy: No history of hay fever or asthma.

Teeth: Most of them out.

Diet: Apparently deficient in vitamins A and B. Gets plenty of vegetables and meat, and a generous amount of starch. Has occasional leafy salads, no milk, and few citrus fruits, but has tomato juice very frequently.

Operations: Tonsils out.

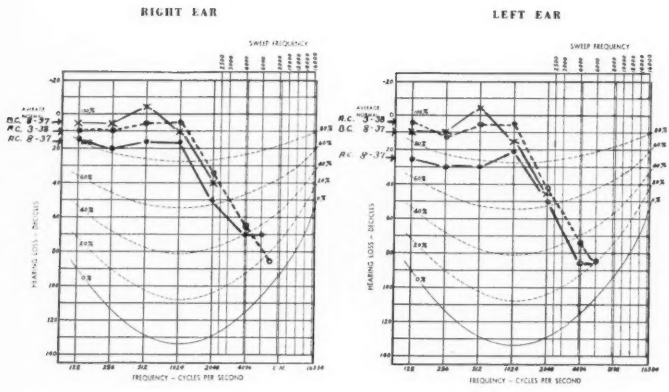

Fig. 6. Case 4

Examination—Physical: General health is good. No colds since the removal of his tonsils. No constipation. Has used tobacco perhaps excessively all his life. No drugs.

Ears: Normal.

Nose: Deviated septum. Taste and smell have lessened.

Tonsils: Out.

Laboratory Data—B. M. R.: (Not done).

Blood Count: Hgb., 92 per cent; RBC, 4,650,000; WBC, 10,700.

Vitamin A: (Photometer reading). Moderate excess.

Blood Analysis: Calcium, phosphorus, potassium, sodium, serum magnesium, and vitamin C are normal. RBC magnesium and cholesterol are high.

Treatment—April 27, 1937: Rx rice bran B complex (used to August 28, 1937).

August 28, 1937: Given injections of riboflavin three times a week (to October 28, 1937).

October 28, 1937: Given liver extract injections once a week because it contained vitamin B_1 and the filtrate factor (to December 10, 1937).

December 29, 1937: All previous medication was stopped and nicotinic acid capsules were given (60 mgm. daily) to see if under its use the audiometric curve would step up further.

March 16, 1938: Dose of nicotinic acid capsules increased to 0.12 gm. daily. (Has taken this to present date).

Comments: This case is the only one who apparently showed any beneficial effects from the use of riboflavin. On October 13, 1937, this patient made the following comments: "I am now able to sleep right straight through the night. My sense of smell and taste have greatly improved, and there has been a great

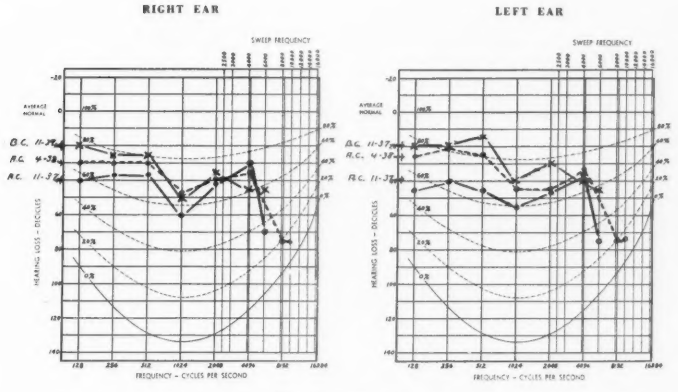

Fig. 7. Case 5

improvement in my hearing. I no longer have palpitations of the heart at night when I lie down, and the very noticeable tremors of my fingers have disappeared. I feel better in every way!"

Case 5.—Mrs. H. K., age 62; December 29, 1937.

Complaints—Deafness: Both ears.

History—Deafness: Has tinnitus constantly which is a high-pitched musical sound.

Diet: Diet has been very poor. Far too much carbohydrates and an inadequate vitamin B intake.

Examination—Physical: Done by her family physician, who states there is nothing physically wrong with this patient other than an arthritis and a very poor diet.

B. P.: 143/80.

Laboratory Data—B. M. R.: Minus 13 per cent.

Blood Count: Hgb., 92 per cent; RBC, 4,950,000; WBC, 8,300.

Treatment—December 29, 1937: To take nicotinic acid capsules, 60 mgm. daily, and is to continue taking thyroid, 30 mgm. daily.

Comments: This patient's improvement was quite noticeable during the four months she was under observation, so much so that she was able to hear the telephone bell and speech very much better than before treatment.

Case 6.—Mr. J. B., age 65; April 30, 1938.

Complaints—Deafness: Both ears.

History—Deafness: First noticed deafness in right ear at age of 40. In 1928 he had an attack of vertigo with staggering when he walked and was sick at his

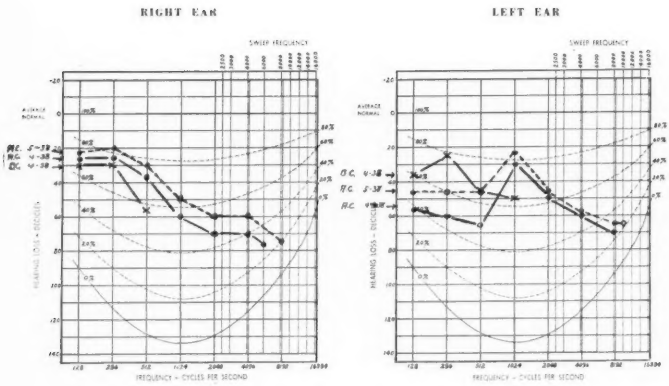

Fig. 8. Case 6

stomach. At the time he was first seen, he stated he was confused when several people were talking. He heard fairly well over the telephone. Patient has no tinnitus. One sister is deaf; hearing of father and mother was good.

Allergy: Has frequent colds, possibly allergic.

Diet: Definitely low in vitamins A, B and C. Has little milk, few eggs, infrequent salads, occasional citrus fruits, but plenty of vegetables and fruits in season.

Operations: Tonsils removed several years ago and also an intranasal operation on the right antrum. (Relief from the antrum lasted one year with frequent recurrent infections, probably allergic in character).

Examination—Physical: His physician reports a low grade prostititis, a variable blood pressure (150 to 200), the usual amount of arteriosclerosis, some slight evidence of cardiac damage, a somewhat unstable vasomotor system, and constipation for years.

Weight: One hundred eighty-three pounds.

Height: Six feet, one inch.

Ears: Normal.

Tubes: Open.

Nose: Normal.

Throat: Normal; postnasal discharge, occasionally yellow in color.

Tonsils: Out.

Hearing Tests: Whisper (R) 3 inches, (L) 4 inches; conversation (R) 18 feet, (L) 24 feet.

Laboratory Data—B. M. R.: Minus 11.5 per cent.

Blood Count: Hgb., 91 per cent; RBC, 5,310,000; WBC, 5,800; eosinophiles, one per cent.

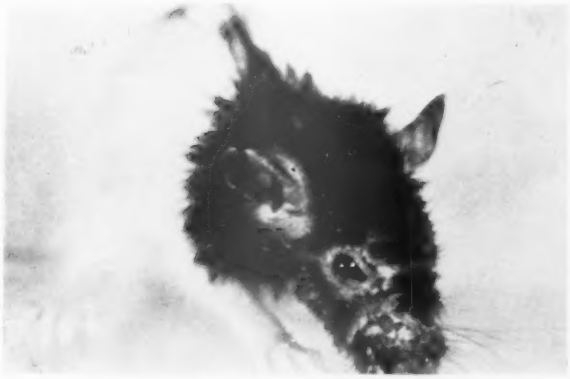

Fig. 9. B_6 rat. Halliday[8] has recently described acute yellow atrophy in these cases. Our animals showed fairly extensive degeneration of the eighth nerve.

Treatment—May 1, 1938: Given nicotinic acid, 60 mgm., t.i.d. (to present date).

Comments: Constipation is almost entirely relieved and there is definite improvement in hearing and general health (November 1, 1938).

SUMMARY AND CONCLUSIONS

The entire story regarding the significance of the various fractions of the B_2 complex has not yet been written, and the relationship of degeneration of the peripheral nerves and general nervous system to the different vitamins, especially the present known fractions of the B_2 complex, has not been completed. Indeed, the story is desperately in need of rewriting!

The gray hair fraction of Morgan and the recently discovered liver fraction of Gyorgy[10] (chirrosis preventative) will singly require histologic study of the eighth nerve, and the clinical trial of these substances singly, if and when they are synthesized, before a full answer can be given as to the real importance of the different fractions in relation to degeneration (senescence) of the nervous system, and particularly the auditory nerve.

At present, nicotinic acid, nicotinamide, and sodium nicotinate appear to be most important.

Fig. 10. Morgan's[9] gray haired rat.

The cases herein reported suggest the importance of determining what fractions of the B complex are most important. Such a procedure was not possible when I wrote my first paper "Eighth Nerve High Tone Deafness from a Nutritional Standpoint"[11] in which I used the whole complex made from California Rice Bran. The present investigation indicates that all the preparations at present on the market do not contain sufficient units of some of the fractions.

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VI

ALLERGY IN OTOLARYNGOLOGY AND ITS RELATION TO OTHER MANIFESTATIONS*

I. General Considerations

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Allergy in its various manifestations may involve a variety of structures in the eye, the ear, the respiratory tract, the upper digestive tract and contiguous tissues. The frequent occurrence of allergy of the nose and paranasal sinuses particularly, constitutes an ever present problem for the otolaryngologist in diagnosis and treatment. Wherever the lesion of allergy occurs, it is characterized pathologically by edema and eosinophilic infiltration, sometimes complicated by infection. Ulceration and hemorrhagic lesions in the form of purpura may also occur. Wherever smooth muscle is involved, spasm results. Involvement of the peripheral nerves may cause paresis or paralysis. It is the edema, however, which constitutes the principal lesion, especially in the respiratory tract.

The diagnosis and treatment of allergy of all types must be based upon the consideration of the following: (1) The shock organs involved; (2) the etiologic factors; (3) the relation of the various manifestations of allergy to each other.

The involvement of the various shock organs and their individual structures with the various allergic manifestations may be enumerated as follows:

The Eye:

Peri-orbital tissues—edema.
Skin of eyelids and lid margains—urticaria, angioneurotic edema, contact dermatitis.
Conjunctiva—allergic conjunctivitis.
Cornea—allergic keratitis.
Uveal tract—uveitis, cataract.

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Retina and choroid—edema with loss of vision.
Optic nerve—edema with loss of vision.

The Ear:

External canal—eczema, urticaria, purpura, contact dermatitis.
Eustachian tube—edema.
Middle ear—edema?
Internal ear—vertigo, tinnitus, deafness.
Auditory nerve—vertigo, tinnitus, deafness.

The Respiratory Tract:

The nose—perennial nasal allergy and hay fever, hyperplasia, polyposis, edema.
The paranasal sinuses—hyperplasia, polyposis, edema.
The mouth—cheilitis, canker sores, stomatitis, gingivitis, glossitis, urticaria, angioneurotic edema, purpura.
The pharynx, nasopharynx, larynx—uticaria, purpura, ulceration, angioneurotic edema.
The trachea—allergic tracheitis, urticaria, angioneurotic edema, purpura.
The bronchi—allergic bronchitis, bronchial asthma, bronchospasm, bronchiectasis.

The Gastro-intestinal Tract:

Esophagus—urticaria, angioneurotic edema, purpura, spasm.
Stomach—gastritis, cramps, distention, retention, belching, hemorrhage, spasm, atony.
Gallbladder—colic, spasm.
Small intestine—enteritis, disturbances of motility, spasm, atony, purpura.
Colon—colitis, diarrhea, spastic constipation, dilation of colon, purpura.
Anus—urticaria, angioneurotic edema, eczema.

The Genito-urinary Tract:

Kidney—nephritis, nephrosis, edema, hemorrhage.
Ureter—spasm, edema.
Bladder—cystitis, edema, ulceration, hemorrhage, spasm.
Urethra—urethritis, frequent urination, burning.
Genital organs—female—dysmenorrhea, spasm of uterus and tubes, edema, hemorrhage, spasm.
External genitalia—urticaria, angioneurotic edema, eczema, purpura, contact dermatitis.

The Skin:

Urticaria, erythema, eczema, angioneurotic edema, purpura, contact dermatitis.

The Bones and Joints:

Bone scorings, disturbances of mineral metabolism, arthritis, edema.

The Nervous System—Sensory, Motor, Involuntary:

Central nervous system—allergic headache, mental confusion, asthenia, irritability, dizziness, epilepsy.
The spinal cord—myelitis, paralysis.
The peripheral and cranial nerves—paresis, paralysis.

The Blood Vascular System:

Hypotension, hypertension, Ménière's syndrome, epilepsy,

At the outset, it is well to tabulate the various causes of clinical allergy which are classified by Rowe[1] as follows:

1. *Inhalants:*

Pollens	Animal emanations
Orris root	Silk
Pyrethrum	Cottonseed
Kapok	Miscellaneous cosmetics and other substances
Hay and feed dusts	Occupational dusts
House dusts	Flours and other dry foods
Recreational dusts	Fly and insect emanations

2. *Ingestants:*

Foods	Condiments
Beverages	Drugs
Dentifrices and mouth washes	Water (mineral and organic content)

3. *Contactants:*

Environmental	Occupational
Medicinal	Cosmetic

4. *Bacteria, fungi, parasites, and products of diseased tissues.*
5. *Injectants:*
   a. Subcutaneous or intramuscular

Sera	Allergens
Drugs	Vaccines
Insect bites	Hormones

b. Intravenous

Sera	Allergens
Drugs	Vaccines
Foods (through transfusion)	

c. Rectally

Drugs	Foods

d. Nasal, aural, sinus, vaginal, urological, intrabronchial, cerebrospinal injections of drugs and allergens.

6. *Allergens transmitted through the placenta to the fetus.*

7. *Physical Allergy:*

Either primary or secondary in action.

Before considering the various types and manisfestations of allergy, it may be well to discuss the matter of terminology in order to avoid confusion. A simplified definition for allergy, as used in clinical medicine today, according to Rowe,[1] may be stated as specific, acquired reaction altered from the normal, arising to a substance which usually produces no physico-chemical or immunologic disturbances in the cells of the tissues. A more simplified definition of allergy may be expressed in terms of "tolerance." It may be said that every person has a "threshold of tolerance" which is individual. When this threshold of tolerance is overstepped, an intolerance on the part of the body is expressed in the form of an allergic manifestation. The allergic individual or potentially allergic individual has a lower threshold of tolerance than the so-called normal. The occurrence of serum disease in normal individuals is an example of overstepping the threshold of tolerance. In those instances, it is a question of the amount of therapeutic serum injected. The larger the amount the higher the incidence of serum reactions. In allergic individuals receiving therapeutic injections of pollen, duct, vaccines, etc., constitutional reactions may occur from over-dosage or overstepping the threshold of tolerance. Gradually increasing doses of pollen or dust, for example, raise the threshold of tolerance, rather than desensitize. Upon the discontinuance of treatment, tolerance is quickly lost. The average duration of tolerance, therefore, is not more than three or four weeks, after maximum dosage has been reached. In the ordinary allergies, the threshold of tolerance is low to pollens, inhalants and foods.

The various types of allergy may be classified or designated according to the etiologic factors or according to the anatomic site or

type of reactions. The various types of allergy, therefore, may be enumerated in two groups, as follows:

Group 1.

Hay fever or pollen allergy.
Nasal allergy—with or without hay fever.
Paranasal sinus allergy—with or without hay fever.
Allergic bronchitis.
Bronchial asthma.
Gastro-intestinal allergy.
Urogenital allergy.
Skin allergy—urticaria, angioneurotic edema, eczema, purpura.
Allergic headache.

Group 2.

Serum disease or serum allergy.
Drug allergies.
Contact allergy.
Bacterial allergy.
Physical allergy.

Group 1 represents the common types of allergy, also designated as the atopic diseases. These manifestations are usually caused by pollens, inhalants, such as animal emanation, dust, etc., and foods. The skin manifestations of allergy, such as urticaria, angioneurotic edema and purpura, may also involve the mucous membranes of the mouth, pharynx, larynx, trachea and esophagus. In this group the most common types of allergy are included.

The various types of allergy listed in Group 2 tend to exhibit lesions of a certain type sometimes confined to certain anatomic sites. Typical serum disease is manifested by the occurrence of urticaria and angioneurotic edema with joint symptoms. The urticaria and angioneurotic edema are usually confined to the skin but may also involve the mucous membranes, especially of the respiratory and gastrointestinal tracts. Reactions similar to serum disease may occur immediately following the injection of an overdose of pollen or may follow the ingestion of some specific food. The ingestion of drugs may excite similar manifestations. Drugs may also cause respiratory symptoms.

The injection of therapeutic sera may also cause lesions of the nervous system. Paralysis following the injection of antitoxin was

reported by Gangolphe.[2] In a tabulation of 49 cases with neurologic complications, Doyle[3] found the brachial plexes the most frequently involved structure (33 cases). Mason[4] reported involvement of the optic nerve in serum sickness. Paralysis of the facial nerve, and the oculomotor nerve by Valerio[5] have also been noted. Generalized paralysis with bulbar and medullary symptoms were noted by Bourguignon[6] following the injection of purified anti-serum. Involvement of the auditory nerve was reported by Cutter.[7] Unilateral recurrent nerve paralysis has been described by Lavrand,[8] Schauwecker,[9] Roger and Bremond,[10] and by Neffson,[11] and bilateral abductor paralysis was noted in the cases reported by Roger, Prevot and Wahl,[12] and by Imperatori.[13]

Reactions somewhat similar to those following the injection of sera may also follow the ingestion of drugs. The principal manifestations are urticaria and angioneurotic edema which usually involve the skin but may also affect the mucous membranes of the respiratory tract. Nasal symptoms and asthma may, therefore, occur following the ingestion of drugs. Swelling of the eyelids, face and lips, urticaria of the mucosa of the mouth, pharynx, larynx, trachea and esophagus, as well as edema of the larynx may also occur. The occurrence of lesions of the upper respiratory tract in agranulocytosis is well known by the otolaryngologist. Squire and Madison[14] have presented evidence to indicate that acute primary granulocytopenia can be caused by repeated administrations of amidopyrine to persons who have developed allergic or anaphylactoid hypersensitiveness to this drug. In two patients hypersensitiveness was demonstrated by patch testing. Three cases of extensive purpuric lesions of the mouth, palate, pharynx, larynx and esophagus were reported by Watson-Williams[15] following the administration of nevarsenohenzol.

Those manifestations of allergy characterized by urticaria and angioneurotic edema, which may involve the pharynx, larynx, trachea and esophagus, are not infrequently encountered by the otolaryngologist.[16] These lesions may or may not be associated with those of the same type occurring in the skin. Many of these cases reported in the literature are designated as idiopathic types. Since they not infrequently occur as a result of food or drug allergy, this etiologic possibility was no doubt overlooked. In many instances the lesions followed the injection of therapeutic sera. In 1905, Halsted[17] reported three cases of edema of the pharynx and larynx of the allergic type. Cases of angioneurotic edema of the larynx have also been reported by Woodbury,[18] Hallock,[19] Lederman,[20] Taylor,[21] Vallery-Radot and Balmoutier,[22] Chittenden,[23] Ballenger,[24] Osler,[25] Griffith,[26]

and Hansel.[27] Among 83 cases of angioneurotic edema observed by Collins,[28] there were six in which the larynx was involved. Angioneurotic edema of the larynx following the administration of therapeutic serum has been reported by Theisen.[29]

Freudenthal,[30] in 1898, reported a case of urticaria of the larynx and later, in 1924,[31] a case in which the lesions were confined to the trachea. Urticaria of the larynx was also noted by Cohen[32] and by Lewis.[33] In Lewis' case the urticaria followed the injection of a pollen extract. Sequi[34] noted urticaria of the larynx following the administration of potassium iodide.

Angioneurotic edema and urticaria of the esophagus was reported by Jackson.[35] In one case the palate and tongue as well as the esophagus were involved. A patient with urticaria of the esophagus also had similar lesions of the skin. In a case of serum disease, urticaria of the esophagus was associated with similar lesions in the skin and swelling of the tongue.

Contact allergy is most frequently observed as contact dermatitis. This type of allergy is one in which only the skin or mucosa is involved. There are usually no other manifestations of allergy present and skin reactions by the scratch and intracutaneous methods are negative. Positive reactions may be elicited by the patch test method. This type of dermatitis may involve the auricle and external canal, the eyelids and lid margins. It has been suggested by Forman[36] that contact allergy may also involve the nasal mucosa, the conjunctiva and cornea. Definite causes of contact allergy of the lids and cornea have been attributed to the use of drugs in this region. No doubt many causes of conjunctivitis in which etiologic factor is not evident are the result of contact allergy.

The part played by bacteria as a cause of allergy of the nose and paranasal sinuses has not been extensively studied. That bacterial allergy may at times produce bronchial asthma or other allergic manifestations has been suggested by Rackemann,[37] Rowe,[38] and others. Rackemann states that bacterial asthma is an example of the late anaphylactic phase of incomplete immunity, characterized by the delayed type of skin reaction. Walker[39] assumed that most patients who failed to give skin reactions to foods and inhalants were sensitive to bacteria. Cooke[40, 41] and also Brown[42] have stated that bacterial allergy is not infrequently a cause of asthma. Forman[43] has demonstrated bacterial asthma associated with immediate wheal activity. Rackemann classifies those cases of negative skin reactions as intrinsic asthmatics. Cohen and Rudolph[44] have suggested that

bacterial allergy is frequently diagnosed because skin reactions to other allergens are not demonstrable. Many allergists, however, feel that many of the intrinsic asthmatics are really sensitive to inhalant or ingestant allergens. Some doubt as to the frequency of bacterial allergy arises because of the unsatisfactory results obtained from vaccine therapy and following the surgical removal of foci of infection. Many allergists, therefore, doubt the occurrence of actual asthma primarily and solely due to bacterial allergy. Because of the fact that attacks of bronchial asthma are not infrequently preceded by acute infections, has suggested bacterial allergy as a cause. Rowe[45] suggests that infections may lower the allergic threshold so that underlying potential inhalant or ingestant allergy becomes active and bronchial asthma results. In such cases bacterial infection, therefore, is secondary and not an allergic cause. In patients with nasal allergy, acute edematous polyps may result from acute infection. In these instances the polyposis is inflammatory and not allergic in nature. No doubt similar inflammatory swelling occur in the bronchi in asthma due to acute infection.

Such observers as Huber and Koessler,[46] Feinberg,[47] Knott,[48] Cohen and Rudolph,[44] Bray,[49] and others have found that typical allergy to bacteria is rare in asthma. Kern and Donnelly[50] have shown that sinus infection is a secondary result and not a primary cause of asthma. Among 400 cases of sinusitis, Bullen[51] found asthma in only 12.5 per cent. Cooke and Grove[52] concluded that sinusitis was an etiologic factor in 92 per cent of 248 selected cases of so-called infective asthma. Grove and Cooke[53] state that the thickened membrane of chronic hyperplastic sinusitis is due to infection and not to a reaction to inhalant substances or foods. From these reports it is difficult to determine the actual incidence of bronchial asthma arising from bacterial allergy. Upon the basis of his extensive experience, Rowe[54] feels that bacterial allergy is the fundamental cause of bacterial asthma in a very small percentage of cases.

The literature is replete with reports of the relation of asthma to pathologic changes in the nose and paranasal sinuses. Until only a few years ago the edematous swelling of the mucosa found in the nose and sinuses and the formation of polyps were considered of infectious origin and naturally suggested surgical therapy. It is now generally conceded that no surgery on the nose or sinuses in patients with nasal allergy and asthma should be performed until careful allergic investigation and treatment over a period of several months have been instituted. The results obtained by surgical procedures on the nose and paranasal sinuses in allergy and by numerous observ-

ers have been reported by Rowe,[1] Hansel[55] and Vaughan.[56] An analysis of all these reports shows that the results on the whole were unsatisfactory. Temporary results were often good but could be explained on the basis of trauma and nonspecific reaction. The most favorable benefits in bronchial asthma from nasal surgery have been reported by Grove and Cooke.[52] Among 120 cases of infectious asthma, 247 operations were performed. Definite improvement in 70 per cent was reported. Later Cooke and Grove[53] again reported similar results in a large group of cases.

Hansel[55] has pointed out that the pure eosinophilic response in the secretions is indicative of the occurrence of allergic reactions in the local tissues, and emphasis has been placed upon the fact that acute and chronic complicating infections superimposed upon allergic processes are characterized by neutrophilic responses in the secretions. Cooke[57] is also of the opinion that a pure eosinophilia may occur in the nasal secretions in hyperplastic sinusitis due to bacterial allergy. In cases of hyperplastic nasal and sinus disease or those with polyposis, there occurs a certain degree of stagnation of the secretions. The infection, therefore, is a secondary process. The secretions in these cases often show a considerable number of neutrophiles which usually disappear from allergic management and restoration of ventilation and drainage of the sinuses. In some instances surgical removal of polypoid tissue is necessary to establish this ventilation and drainage. It has already been pointed out that edema and polyposis may be produced by an acute or chronic inflammatory process in an allergic individual, but in these instances we have repeatedly demonstrated that the cytologic response of the secretions is predominantly neutrophilic and not eosinophilic. In general, we have found that pure bacterial allergy is a very rare cause of edematous changes in the nose and paranasal sinuses.

Physical allergy may be defined as that type of abnormal response to environmental agents such as heat, cold and sunlight. The symptoms, signs, diagnostic criteria, and tests in physical allergy have been most thoroughly studied by Duke.[58] The clinical aspects of his investigation have been confirmed by a number of observers. Spiesman[59] has shown that the nasal mucosa in nasal allergy responds erratically to moderate thermal stimuli, whereas the infected mucosa does not respond at all. Peters and Hoffman[60] noted that during attacks of hay fever and asthma, heat produces a decrease in body temperature and ice rubs cause a rise in temperature. When the same patients were free of symptoms, the heat would raise the temperature and the ice would lower it. Duke has shown that the mani-

festations of physical allergy are due to a disturbance of the heat regulating mechanism.

In a clinical investigation of the part played by physical allergy, Swineford[61] pointed out the following significant findings: In a group of 325 patients, 201 attributed an appreciable amount of their trouble to physical agents, more often than to any one group of material agents. In the entire group, 14 symptoms attributed to 16 normal environmental influences occurred 629 times. In 63 cases, by simple physical tests the chief complaint was reproduced appreciably 41 times, and other symptoms 11 times. Nine symptoms were relieved promptly by the application of the physical (thermal) influence opposite to that which produced them. In the above 63 cases, nasal congestion occurred 20 times; cough, 10; wheezing, 14; headache, 7; urticaria, 4; abdominal cramps, 4, and angioneurotic edema, eczema and hoarseness once each, a total of 62 symptoms in 63 tests. The effective test was infra-red in 19, ice in 36, both simultaneously in 4, ultraviolet in 3.

With the exception of acute infections, allergy is the most common affection of the nose and paranasal sinuses. The diagnosis of allergy, therefore, is a problem which concerns the rhinologist in his daily practice. In order to insure accuracy in diagnosis, consideration must be given to the following: (1) The nasal and sinus symptoms; (2) the rhinologic examination; (3) the cytology of the secretion; (4) the roentgenographic examination, and in certain cases (5) the histopathologic and (6) the bacteriologic findings. In sinus diagnosis, aspiration and lavage may be indicated to produce secretions for cytologic and bacteriologic examinations. If the information obtained from this routine investigation is properly correlated, diagnosis can be established with the greatest degree of accuracy.

Since the common cold is an ever-present possibility, it may temporarily obscure the allergic picture. Repeated observations may be necessary, therefore, to determine whether the infection is acute, subacute, or possibly chronic.

The symptomatology of nasal allergy is characterized by sneezing, itching, obstruction and discharge. In typical cases these symptoms are so classical that the diagnosis is perfectly evident. In atypical cases, however, the symptoms may be intermittent, simulating recurring attacks of acute rhinitis, or there may be an absence of sneezing and itching. Obstruction and discharge in varying degrees may be the only symptoms, in which instances, allergy must be differentiated from other conditions which are characterized by similar

complaints. These atypical cases of allergy are the ones which are most commonly overlooked.

Upon rhinoscopic examination, the most typical changes in nasal allergy are characterized by intumescence and pallor of the mucosa. In some instances edema varying from a slight swelling of the mucosa in the middle meatus to marked polyposis may be noted. There are many cases, on the other hand, in which pallor and edema are absent. The mucosa may appear normal in color or even slightly reddened. A normal appearing mucosa is likely to be present during periods of quiescence of symptoms, and this is especially true in those patients with intermittent reactions. Marked acute exacerbations of allergy may produce a temporary acute edema in the middle meatus or even acute edematous polyps which disappear upon the subsidence of symptoms. Acute infections in nasal allergy may also produce an inflammatory edema or acute polyposis which disappears when the acute infection subsides.

The edematous process which occurs in the nasal and sinus mucosa is somewhat comparable to that which appears in the skin in urticaria. In the skin an urticaria wheal appears and within a short period of time it disappears and another wheal may appear elsewhere. The edematous reactions, therefore, are of short duration and the edema readily absorbs. This is also true in the mucosa of the nose and sinuses. An acute allergic reaction produces an edema in the mucosa which is visible as pallor. When the acute reaction subsides the edema absorbs and the pallor disappears, thus accounting for a normal appearing mucosa in many instances during a quiescent period of symptoms. In some cases an acute allergic reaction may produce an acute polyposis. This is especially common in hay fever. An acute inflammatory edema may also result from an acute infection. Here the cytologic picture of the secretions may be the only differentiating factor. Acute edematous reactions may also occur in the paranasal sinuses and disappear within 24 to 48 hours. This has been repeatedly demonstrated by means of the x-ray. In cases of chronic polyposis there is a tendency for the polyps to vary in size according to the degree of allergic activity. During a period of marked reactions, the polyps become enlarged, usually appearing very pale and boggy. During an interval of quiescence they may be much smaller, less pale and more compact. Repeated observations, therefore, are necessary to determine to what extent polyps are involved in an acute process. If fibrosis becomes established and the edema becomes fixed, there may be little variation in their size, regardless of the allergic activity. If, after a period of observation

and allergic therapy, obstructing polyps remain, they should be removed. Sinus surgery should always be deferred until nasal respiration and ventilation are restored. Sinus edema may subside under allergic treatment. Stagnation and retention of secretions with secondary infection may also disappear. Cytologic studies of sinus and nasal secretions should be repeatedly evaluated. If marked sinus edema or polyposis with infection persists, surgical methods are indicated.

From the standpoint of symptoms and nasal findings, diagnosis in many instances is purely a matter of conjecture, but this can be eliminated by determining the cytologic response in the secretions.

Wherever the lesion of allergy occurs, it is characterized by edema and eosinophilic infiltration. The edema in the nasal mucosa is noted as pallor or as polyposis. The eosinophiles are found in the secretions upon microscopic examination, even when there may be no visible evidence of edema. The presence of eosinophiles in significant numbers is absolute evidence of the allergic process. A persistent absence of eosinophiles, except in cases of marked complicating suppuration, definitely excludes the possibility of allergy. During acute infections the secretions may contain only neutrophiles and a few scattered or no eosinophiles. As the acute infection subsides, the neutrophiles gradually disappear and the eosinophiles gradually reappear in increasing numbers, the secretions finally becoming clear and watery or mucoid. Upon the occurrence of an acute exacerbation of nasal symptoms, it may be a question of whether the reaction is allergic or an acute infection. While the secretion is still clear and mucoid, the microscopic examination of the secretions may be the only means of differentiation, assuming that acute sore throat and constitutional symptoms may be indefinite. By repeated cytologic observations over a prolonged period of time, it is possible to determine the exact incidence of acute infections in the allergic individual.

With marked nasal obstruction, secondary infection of the stagnation type may occur, in which the secretions show a considerable number of neutrophiles as well as eosinophiles. Upon restoration of nasal respiration the neutrophiles quickly disappear. In acute infections the neutrophiles persist in the secretions during the entire period of duration of the acute infection (10 or 15 days). In cases of nasal polyposis, neutrophiles are always present with the eosinophiles because of a constant stagnation process. After removal of the polyps along with the control of the allergic reactions, the neutrophilic stag-

nation process tends to disappear. On the basis of these observations it is evident that the cytologic picture of the nasal and sinus secretions is of indispensable value as a guide in the diagnosis and the clinical course of the symptoms in relation to infection.

Since the patient with nasal allergy has become accustomed to the constant presence of nasal discharge, he is often not aware of the fact that an exacerbation of symptoms is caused by an acute infection. If fever, sore throat and a definite purulent discharge are present, the infectious process is evident. On the other hand, the more mild acute infections may not be accompanied by these symptoms. The discharge may not be purulent. On account of the profuseness of discharge in allergic patients, there is a high dilution of the cellular elements in the secretions, hence they may not appear grossly purulent. Microscopic examination, however, reveals a very large number of neutrophiles. In general, the gross appearance of nasal secretions may be no index of the type of cellular elements present.

An x-ray examination of the paranasal sinuses, to determine the possible chronicity of the allergic changes, should not be made during an acute exacerbation of symptoms, whether caused by the allergic process or by an acute infection. Under these circumstances there is an acute edematous process which is very likely to show marked changes in the x-ray plate, and which may be temporary rather than permanent. The chronicity of the changes are better evaluated after allergic management has been instituted. In any event, the x-ray findings should always be correlated with symptomatology, cytology of secretions, local changes in the nose, and bacteriologic examinations.

A histopathologic examination of all tissues removed from the nose and paranasal sinuses should always be made routinely. In nonsurgical cases a biopsy of the tissues is usually not necessary, because the cytologic picture of the secretions is a reliable index of the pathologic process present.

All bacteriologic findings should be correlated with the cytology of the secretions, at the same time taking into consideration the symptomatology, the gross pathologic changes, and the x-ray findings. In the usual cases of nasal allergy without evident polyposis and a pure eosinophilia of the secretions, bacteriologic findings are usually not significant. When pathogenic infection is present, there is always a large number of neutrophiles in the secretions. In acute infections, virulent bacteria may be cultured from the secretions. In

cases of nasal polyposis with sinus edema and polyposis, the presence of bacteria may be associated with an acute, a subacute or a chronic process. In chronic conditions they may be associated with the stagnation process or with a true pathogenic suppurative involvement. In the stagnation process the infection is usually of the saphrophytic type and disappears with the control of allergic reactions and the restoration of ventilation and drainage. In these instances the cytologic study of the secretions is of the most significance in diagnosis. In the chronic true pathogenic complicating suppurations, the secretion is markedly purulent with almost all neutrophiles present. The bacteria present are more virulent, constitutional evidence of infection is usually present and the local condition usually does not show satisfactory response to allergic management. In our experience we have found that only a small percentage of cases are classified in this group.

From the foregoing discussion of routine investigation, it is apparent that accurate diagnosis is dependent upon a correlation of all data obtained. Although the cytologic study of the nasal secretions not infrequently establishes the diagnosis, the symptoms and nasal changes may be indefinite or atypical. Such symptoms as sneezing and itching, when persistent, are typical of allergy, but nasal obstruction and discharges are commonly present in non-allergic conditions. Vascular and secretory disturbances in the nose may be present in such conditions as acute and chronic rhinitis, cerebrospinal rhinorrhea, chemical and nonspecific dust irritations, hypertrophic rhinitis, atopic rhinitis, syphilis and tuberculosis. Such disturbances may also occur secondary to disease elsewhere, such as endocrine disease, avitaminosis, circulatory and kidney diseases.

In the presence of typical edematous changes in the nose, characterized by hyperplasia and polyposis, there is visible evidence of a probable allergic process. In the absence of typical changes, the possibility of allergy can be easily overlooked. During an acute head cold the infectious process may temporarily obscure a chronic nasal allergy.

In the diagnosis and treatment of respiratory allergy, the possibility of seasonal hay fever must always be taken into consideration. In most localities three distinct hay fever seasons are recognized; namely, the tree, grass and ragweed seasons. Among those patients who have hay fever, some have symptoms only during the specific pollinating seasons, while some others have perennial nasal allergy in addition to the hay fever. Tree and grass hay fever are usually less severe and may be more intermittent than the ragweed type. Tree

and grass hay fever may be considered as seasonal head colds and may, therefore, be overlooked. Patients with perennial nasal allergy who also have hay fever exhibit distinct exacerbations of symptoms corresponding to the season of pollination of the plants to the pollen of which sensitiveness is present. Among those patients whose nasal allergy consists only of hay fever, it is important to note whether other manifestations of allergy, such as skin allergy, gastrointestinal allergy, or allergic headache also occur during the hay fever season or at other times during the year.

Chronic bronchial irritation, characterized by cough and the expectoration of mucus, may be a manifestation of allergy. Cooke[62] has called attention to the occurrence of a paroxysmal type of cough in children which may simulate pertussis. Cough is often a forerunner to asthma in children. It usually accompanies nasal allergy. Duke[63] emphasizes the importance of differentiating between allergic bronchitis and pulmonary tuberculosis, chronic bronchitis or bronchiectasis. The cytology of the bronchial secretions is of indispensable value in establishing the diagnosis. The symptoms are relieved by ephedrine or epinephrine. Nasal allergy is almost always present with allergic bronchitis. It may be very mild, hence easily overlooked. The importance of recognizing allergic bronchitis has also been emphasized by Rowe,[64] Colmes,[65] Colmes and Rackemann,[66] Kahn,[67] Ratner,[68] Brown,[69] Waldbott[70] and Waters.[71]

Correctness in the diagnosis of asthma is always important. It is practically always accompanied by nasal allergy and the symptoms are generally relieved by ephedrine or epinephrine. The cytologic studies of the nasal and bronchial secretions is obviously important. The possibility of an acute infection should also always be taken into consideration. On the other hand, Jackson's[72] dictum that 'all that wheezes is not bronchial asthma" must be remembered. All the lesions involving the structures from the pharynx and larynx to the terminal bronchia and lungs which may produce dyspnea and wheezing and which may, therefore, simulate asthma, must be taken into consideration. Among these various conditions, the following may be enumerated: pharyngeal abscess or edema, angioneurotic edema, spasm, paralysis or tumors and foreign bodies in the larynx, tracheobronchial stenosis from edema, tumors or foreign bodies, compression of the trachea from tumors, intrathoracic goitre, enlarged tracheobronchial glands or lesions in the esophagus. Various diseases of the lungs may also cause dyspnea simulating asthma. Cardiac and circulatory diseases, kidney diseases, nervous and metabolic diseases, may also cause dypsnea.

SUMMARY

1. Allergy in its various manifestations may involve practically any of the organs and tissues of the body. A great variety of structures may also be involved in the head and neck which concern the otolaryngologist. Those manifestations which occur in the head and neck may be associated, therefore, with those which occur simultaneously elsewhere in the body.

2. Allergy of the nose and paranasal sinuses presents a problem of primary importance to the otolaryngologist.

3. Positive diagnosis should carefully consider the following factors: (a) The nasal and sinus symptoms; (b) the gross pathologic changes; (c) the cytology of the secretions; (d) x-rays of the sinuses; (e) bacteriologic findings; (f) histopathologic findings. All these factors should be correlated in diagnosis.

4. The part played by acute, subacute and chronic infections should be evaluated.

5. The occurrence of other manifestations of allergy, such as hay fever, asthma, skin allergy, gastrointestinal allergy and allergic headache, should be considered in their relationship to allergy in the nose and paranasal sinuses.

6. Such specific types of allergy as contact dermatitis, serum disease, urticaria, angioneurotic edema, eczema and purpura, may involve the skin and mucous membranes of the ear, nose and throat and contiguous structures. These types of allergy may occur as individual manifestations involving one or more of these tissues or may be associated with similar lesions in distant structures of the body.

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VII

NON-SURGICAL TREATMENT OF DISEASES OF THE NOSE AND PHARYNX*

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In preparation for this paper I read over most of what has been written on treatment of the nose and throat during the past five years. Of course, this included surgery which is not within the scope of this paper, yet all of it made interesting reading. If one separates the chaff from the wheat, a very good picture can be seen as to the proper way to treat rhinolaryngologic diseases. However, a discerning mind must be put to the task or one will find himself treading paths in unknown fields. The extremes of the picture furnished the most interesting, if not trustworthy, information. As an example, one method of treatment was advocated as a valuable remedy in the relief of acute sinusitis. I have forgotten the name of the author and even his method, but I recall his closing sentences. In these he advocated the use of his procedure and used as an argument in its favor, the fact that most of his acute cases recovered within a month. One wonders if his treatment did not prolong the condition, to make them last that long. At the other end of the scale, some of our surgically-minded friends assure us that success is ours if we will only place a certain flap of mucous membrane in a certain place, or do some equally exact maneuver. I cite these instances, not to ridicule the authors, but to emphasize the difference in viewpoint with which this problem is approached. One must know the background and work of the author before an article on treatment can be properly appraised.

Not only is this variation in viewpoint confusing, but there are several other factors which make it difficult for a student of therapeutics to know the truth about the subject. Difference of opinion between two or more doctors about the proper treatment of a certain condition may arise from the fact that while they may use the same drugs, their techniques are not alike. Further, the difficulty of using controls in many of our treatments makes for reported results

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which seem at variance. Occasionally it is possible to use specimens of tissue or cultures, which will, in a reasonable measure, check the results. Where this is not done, the variation of individuals within a treated group can make for confusing conclusions. Patients taken from different strata of society, and different locations, make for greater variations.

Perhaps the greatest single cause for discrepancies is errors in diagnosis, or, if not actual errors, there is failure to allow for contributing causes which are great enough to invalidate any report. These are matters which are too obvious to need elaboration.

Finally, the most complex factor of all comes into the picture, i.e., personality. This is dual, for it includes the personality of the patient as well as that of the doctor. In treatment, the effect of the enthusiasm of the doctor on the mind of the patient must be taken into consideration. In my student days, a certain procedure was in vogue. Its staunchest advocate was a man of dominant personality. He wrote thirty-five articles on the subject. He was convinced of its worth and, I think, convinced his patients of its worth. I found the subject mentioned but three times in the literature of the past five years. I cite this, not to discredit the doctor, for I know he had ability, but to point out that not a small part of our success as physicians lies in the fact that our patients believe in us and sometimes get well, not because we treat them in a certain way, but in spite of it.

Then there is the effect of the patient's personality on the doctor. In our position, we are often placed on a pedestal (and almost as often it is undeserved), and looked up to by an admiring clientele. It is easy for us to believe we accomplish more than we do. To all of us, some more than others, come patients with an imagined illness, and if we are not careful we can believe we are curing these folks. I admit this problem is one of more moment to the general physician, even more so to the gynecologist, but the otolaryngologist has his share. It may be that I am different from the rest of you in recognizing this as a problem. I don't think so. I am just trying to do a little honest thinking.

With these pitfalls in mind, let us proceed to a discussion of some of the methods used in the non-surgical treatment of the nose and pharynx. Monson[1] has advocated the use of sclerosing solutions in epistaxis. He injects 0.1 cc. of a saturated solution of quinine lactate in four or five places in the nasal mucosa about the site of the bleeding. I have had no experience with this method. Cautery, whether actual or chemical, is efficacious.

For acute rhinits, there are nearly as many methods of treatment as there are doctors who treat the condition. I know of no drug or measure which will consistently shorten the duration of colds. It is true that occasionally we treat what appears to be an acute rhinitis and in twelve to twenty-four hours the patient is rid of his symptoms. It is probable that these attacks are the result of allergy, dietary indiscretions, etc. To enumerate everything which has been suggested or used for acute rhinitis would require several hours for the recitation. A few words, however, can be said about some commonly used measures. Rest and elimination need no emphasis except perhaps to say that it is generally conceded that the former is the most valuable remedial agent we have for acute infections of the nose and throat. If there is no fever, it is apt to be neglected. It is rather common practice to prescribe a vaso-constrictor for acute rhinitis. Its use is, of course, not curative but often gives temporary relief. The work done by Lierle and Moore[2] and Proetz,[3] which was confirmed by Walsh and Cannon,[4] on the action of certain drugs which are used in the nose, is of value to all otolaryngologists who use the information. Working independently, they come to the conclusion that most drugs used in the nose have a depressor action on the cilia of the nasal mucosa. The three most commonly used vaso-constrictors—ephedrine, epinephrine, and cocain—do not fall in this class if they are of the proper strength. Cocain solutions up to five per cent, ephedrine to three per cent, and weak solutions of epinephrine in physiological saline, have no harmful effects on the cilia. Addition of other ingredients detracts from their efficiency.

Some new remedies for acute rhinitis have been recommended, among them sulphur dioxide by Rawlins[5] and Camirol, a camphor-mentholiodoform solution advocated by Coates, Davis and Gordon.[6] These are too new to evaluate at the present moment.

The management of acute sinusitis should be essentially medical. Occasionally surgery must be in the picture, but this is usually when there are complications, or threatened complications. Rest, elimination, proper diet, shrinking sprays, heat and medication for pain are in order. The frontal sinus, though not most frequently involved, seems to give the most trouble. I think I have been reasonably successful in the management of the frontal sinusitis cases which have come to me for treatment. I take no credit for this, but I do take pleasure in giving credit where it is due. When I was a student, Doctor Mosher advised one thing which I have remembered. He told us to have respect for the virginity of the naso-frontal duct. It was good advice. I believe the less intranasal manipulation there is

in uncomplicated frontal sinusitis, the more fortunate the immediate and subsequent history will be.

The late Campbell Smyth was another teacher whose advice was valuable. Some time after I had started practice I heard him say that in acute maxillary sinusitis he only occasionally resorted to irrigation. At this time he said that rest in particular, and the usually applied local measures, would bring about a cure in the great majority of cases. I have found that he was right. I find I irrigate the antrum much less frequently than was my former practice. Incidentally, when puncture is indicated, I am one who adheres to the inferior meatus as the site of choice. VanAlyea[7] has reported that of 163 anatomical specimens, the maxillary ostia were inaccessible for middle meatal irrigations in fifty per cent. I have examined a number, not as great as his, and the percentage of inaccessible ostia is nearly the same. If it is inaccessible, and the attempt is made and the antrum entered, trauma must be present. It seems to me that if there must be trauma, it is better to have it at a point some distance from the ostium.

Heat is frequently used in acute sinusitis. It can be applied in several ways. Andrews and Osborne,[8] in a study of the temperature of the maxillary sinus after treatment by various methods, place them in order of efficiency as follows: electromagnetic field and diathermy, thermospectral lamp, Cutler water-cooled lamp, compsolite short-wave diathermy, and the Elliott machine. But, they tell us, the maximum rise in temperature was from 98.2 to 99.1 degrees. If this is true, the benefits of heat in sinusitis do not come from marked changes in the temperature of the sinuses themselves. The electric head bath, not mentioned above, is another method of applying heat. At times it is quite effective. Occasionally, warm physiological saline nasal irrigations, properly given, will afford much relief in acute sinusitis.

To facilitate discussion, I am grouping together chronic rhinitis, hypertrophic rhinitis, vasomotor rhinitis, allergic rhinitis, and certain forms of chronic sinusitis. I am excluding maxillary sinus infections of dental origin and chronic infections of the sinuses which are obviously due to anatomical abnormalities. If these are excluded, my problem with this group of chronic conditions is diagnostic rather than therapeutic. Those who suffer from these ailments have many complaints, but speak chiefly of difficult nasal breathing and postnasal discharge. The correct solution for the relief of these symptoms has been a challenge to me. In the first year or two of my

practice, when I was still enthused with hospital routine where radical surgery on the sinuses is in order, I was puzzled when I examined patients who complained of postnasal discharge and I could find no cause in the nose for the complaint. I asked myself, "How does one treat these cases?" About this time, a rather dramatic incident happened. A patient with profuse postnasal discharge was referred to me by a fellow rhinologist. He had made a diagnosis of chronic sphenoiditis. There was x-ray evidence to confirm his diagnosis. He felt operation was indicated and asked me to do it because he was related to the patient. I was flattered because he had chosen me and hoped to do the operation. When this patient presented herself for examination, I thought her general condition did not warrant surgical interference. She looked ill and was, in fact, much worse than when she had been examined at home. I called for medical consultation. It was soon found that the patient was only one step away from diabetic coma. Proper treatment was begun, and as the diabetes cleared, the postnasal discharge improved. I have been looking for causes other than nasal ever since. I am not always successful in my search for them, but the more diligent I am, the more I find and the better are my results. A detailed account of all the possible general causes for chronic nasal conditions would unduly lengthen this paper. You know them better than I, but they must underlie a large portion of our practice, when such reliable observers as Baum,[9] Hansel and Mullin[10] state that allergy, which is one of the possible general causes, is present in about 30 per cent of all nasal conditions. This figure may be too high or too low, but it gives a hint as to the size of this problem.

The worth of non-surgical treatment of these disorders, as advocated by various authors, is difficult to evaluate. One does not know whether or not general causes have been eliminated. A great many observers, however, including Mithoefer,[11] have stressed the importance of the condition of the host as a contributing factor in chronic nasal conditions, and these reminders are timely.

Diet is important, for diet can condition the host. Some good articles have appeared on the effects of various deficiencies and excesses. The use of specific diets is sometimes urged for the relief of the conditions under discussion. Our task would be easy if it were as simple as that. As I see it, the proper diet for a patient in this group must be decided on the needs of the individual case. Sometimes correction of the diet is the answer to the problem.

Except for suggestions for symptomatic treatment, there have not been many articles during the past five years dealing with treat-

ment for this type of patient. Galloway,[12] Hollender[13] and Bernheimer[14] have reported on the use of physical therapy. The latter reports on the effect of irradiation on allergic nasal mucosa, and states that of forty persons treated, after three years, forty-seven per cent are free from symptoms. I have had no experience with this type of treatment, nor with the present wave of enthusiasm for short-wave diathermy. Further reports will be needed before these remedies can be accepted as valuable agents.

Jarvis[15] has reported on the use of insulin in otolaryngologic practice. I have used insulin but cannot get as favorable results as his.

Ruskin[16] favors the use of nucleic acid in nasal disease, stating there is a loss of nucleic acid from the cells of the mucosa in the nasal discharge.

The treatment of atrophic rhinitis has always been unsatisfactory. The recent work of Blaisdell[17] and others on the use of estrogenic substances in this disease, gives promise of greater relief than we have been able to give in the past.

Passing from the nose to the nasopharynx, treatment of the diseases of this part should be similar to that used in corresponding conditions in the nose. The adenoids are a factor in chronic nasopharyngitis, as are adenoid remains, infected retention cysts and bursitis, but the treatment for all of these is essentially surgical.

Treatment of acute and chronic pharyngitis should be along the same lines. Acute pharyngitis can be cared for by the use of rest, eliminations, etc., as advocated for the nose. Heat, in this locality, can be applied by irrigations with less danger than in the nose. In chronic pharyngitis search should be diligently made for some local or general cause. The chief local cause is, of course, chronic tonsillitis, but there are many general causes. For instance, Lillie[18] has written an article advocating the use of iodides in chronic granular pharyngitis, not to be applied locally, but to be taken internally. If the use of iodides improves a chronic granular pharyngitis, it suggests that there is some disorder in the iodine metabolism. Many other examples could be given, but the point to stress is that we will get better results if we are good diagnosticians than we will if we only apply local remedies.

For acute tonsillitis, whether pharyngeal or lingual, I would rather have rest and elimination as my therapeutic aids than any other measures of which I know. Sulfanilamide is given credit as a

valuable drug in acute tonsillitis, but the vast majority of these cases, in a very few days, are quite well. For certain complications, both here and in the sinuses and ear, sulfanilamide has made a place for itself in our armamentarium. It is, however, a potent drug and ones does not need to administer it in every case of acute infection of the upper respiratory tract. It is well to remember also, that according to latest opinion, it usually does not kill bacteria but merely inhibits their growth. If given early there is some danger if it is stopped too soon. An exacerbation may ensue because the bacteria may still be present and the patient lack enough antibodies to inhibit their growth. It is thought that a delay of a day or so at the start of an infection will give the antibodies an opportunity to develop and then, if the drug is stopped a little early, nature's own defense mechanism will control the infection. In a complication like meningitis, it should undoubtedly be given early and continued for a considerable period.

Many remedies have been offered as a cure for Vincent's angina. The fact that there are so many of them suggests no one is much better than the other. I have often wondered if in the application of various drugs we do not permit oxygen to get to the depths of the ulceration and thus prevent the continued growth of the anaerobic organisms.

The treatment of tuberculosis and syphilis of the nose and throat is fairly standard and does not need discussion here. The same may be said for diphtheria.

I have tried, in presenting this paper, to avoid recommendation of specific remedies for use in certain conditions. This does not mean that I neglect to use some of them in my practice. I have tried rather to stress the need for a proper approach to our therapy and the necessity for straight thinking in reporting results. Probably none of us will know perfection in therapeutics, but continued effort along these lines will bring us nearer to it.

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VIII

DERMATOLOGIC CONDITIONS OF THE EAR, NOSE AND THROAT*

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Dermatologic conditions of the ear, nose and throat may occur entirely as a local process; more commonly, however, they are a manifestation of some systemic disease. The large number of such conditions encountered makes it impossible to discuss all of them in a single paper, and only those of greatest clinical importance or of particular interest for other reasons will be considered here. The neoplasms, the acute exanthemas and some of the rare dermatologic conditions which only infrequently produce manifestations in these regions must of necessity be omitted.

Various systemic diseases and dermatoses are frequently accompanied by lesions of the ears, nose, mouth and throat. Glossitis and fissuring at the angles of the mouth often develop in secondary anemia, and a more or less characteristic atrophic glossitis is seen in pernicious anemia. Sensitive superficial erosions occur about the mouth and tongue in sprue. In the early stages of typhoid fever, submucous hemorrhages are commonly present in the nose and throat while, in the later stages, deep ulcerations may develop in these situations. Victims of pellagra and other vitamin deficiency diseases often have stomatitis and glossitis. Gingivitis and stomatitis (catarrhal) are often present in diabetes. Petechiae, and at times ecchymoses, may appear on the palate in purpura. Uremia may be accompanied by either an exudative or an ulcerative stomatitis.

While many acute infectious conditions occurring about the alveolar processes present a problem that is more distinctly dental than medical, nevertheless the otolaryngologist should be thoroughly familiar with them. The lesions of Vincent's symbiosis, while almost invariably originating about the gingival borders, commonly involve

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other portions of the oral cavity and not infrequently the pharynx as well. A single, or multiple large ulcer on the base of the tongue may, in fact, be the most prominent manifestation of this condition.

The striking resemblance of the clinical picture in Vincent's infection to that at times seen in certain blood dyscrasias, especially acute and chronic lymphatic leukemia, often causes considerable difficulty from the standpoint of differential diagnosis. These conditions may be indistinguishable on the basis of the clinical examination alone, and a definite diagnosis should always be withheld in cases of this type until hemocytologic studies have been made, even though smears from the gingival ulcerations reveal numerous Vincent's organisms.

In leukemia, especially of the lymphatic type, there may be, in addition to diffuse infiltration and ulceration of the gingiva, hypertrophy of the faucial and lingual tonsils, nodular infiltration of the palate or pharynx, or hemorrhagic, bullous, or necrotic lesions in these situations.

Agranulocytosis should also be mentioned in this connection. This disease, of undetermined etiology, is characterized by marked anemia, leukopenia and disappearance of the polymorphonuclear leukoctyes from the blood. An increasing number of cases of this condition have been observed in recent years and the indiscriminate administration of certain coal tar products, especially amidopyrine, appears to have had some etiologic bearing. The usual lesion in agranulocytosis is a large sloughing ulcer, situated in the palate or pharynx which tends to progress. While temporary improvement often occurs, remissions are frequent and the disease is commonly fatal.

The Plummer-Vinson syndrome (simple achlorhydric anemia) should be mentioned in this connection. This condition, which is on a nutritional basis, being due to iron deficiency, usually occurs in women. It is quite[6] common in certain parts of Europe, especially in the Scandinavian countries among the poorer classes. It is characterized by dysphagia, secondary anemia, contraction of the mouth (with atrophy of the lips and fissuring at the angles), stomatitis, atrophic glossitis, atrophy and paleness of the pharyngeal and esophageal mucosa, and flat, brittle, spoon-shaped nails. The mucous membranes of the mouth, pharynx, larynx and esophagus in these cases are very prone to undergo malignant change without previous leukoplakial thickening and multiple carcinomas are not infrequently seen in the mouth, pharynx and postcricoid region.

The infectious granulomas, syphilis, tuberculosis, actinomycosis, blastomycosis, rhinoscleroma, leprosy and yaws frequently produce pathologic changes in the upper respiratory and alimentary tracts. The lesions of primary, secondary and tertiary syphilis may appear about the nose, mouth, pharynx and ears. The primary sore, or chancre, is commonly seen on the borders of the lips; it may also occur in the nose, tongue, or elsewhere inside the mouth and it has been reported on the epiglottis. It is characterized by marked induration and ulceration, and the regional lymphatics are enlarged and tender. The most common manifestations of secondary syphilis in the mouth and throat is the mucous patch, although rashes similar to those occurring on the surface of the body, glossitis and persistent ulceropapular sore throat may occur. Tertiary syphilitic lesions in this region include gummas in various stages, scarring of the palate, pharynx or nasal mucosa, perforation of the palate or nasal septum, or collapse of the nose secondary to gummas, leukoplakia and chronic interstitial glossitis. Tertiary syphilis is a prominent factor in cicatricial stenosis of the nasopharynx. In the larynx, tertiary syphilis may present as gumma, ulceration, perichondritis, scarring or stenosis.

Tuberculosis may occur about the ear, nose and throat, either in its attenuated form, lupus, or as a true acute or chronic tuberculous lesion. Involvement of the nose by Mycobacterium tuberculosis is encountered almost exclusively as lupus; in the pharynx and ear the process may exist either as lupus or as true tuberculosis; in the larynx, chronic tuberculous ulcers most often occur. The author has encountered involvement of the nasopharynx with a combination of tuberculosis and active squamous-cell epithelioma; the two conditions are at times coexistent in the larynx. In the ear, the infection most commonly produces a chronic suppurative otitis media.

Lupus occurs relatively infrequently in the United States as compared to the British Isles and continental European countries. About the face, the process commonly involves the nose, upper lip and lobes of the ears and often there is an extension to the nasal and oral mucous membrane. The characteristic pinkish, so-called apple-jelly nodules which usually first develop on the anterior portion of the septum often give way to ulceration and perforation of this structure. The process may heal in some areas while progressing in others.

Tuberculous lesions of the mouth and pharynx occur infrequently considering the prevalence of tuberculosis. They are most

commonly found among individuals with advanced pulmonary involvement and usually appear as painful, superficial, moth-eaten ulcers which are usually multiple. They may involve any portion of the oral or pharyngeal mucous membrane and are likely to produce very little evidence of inflammatory reaction and induration in the surrounding tissues. In the mouth and pharynx, as well as in the larynx, these ulcers indicate a highly virulent infection, or lowered resistance to the infection on the part of the patient, and, as a rule, justify an unfavorable prognosis. About the jaws, a single, deep, indolent tuberculous ulcer with involvement of the bone at times occurs. Direct scrapings from the oral and pharyngeal ulcers will occasionally permit of demonstrating tubercle bacilli and thus of establishing a positive diagnosis.

Solitary tuberculoma may be encountered as a discrete tumor in the body of the tongue, lips, or cheeks, developing insidiously and painlessly. These present the characteristic histologic picture of tuberculosis, but their exact relation to pulmonary tuberculosis is indefinite. Roentgenograms of the thorax in cases of this type observed personally showed evidence of pulmonary involvement, but there was no sign of activity either clinically or roentgenographically. The histologic picture of gumma is practically indistinguishable from that of tuberculoma; in all of these cases, however, the serologic findings were negative. Excision was at times followed by recurrence, whereas irradiation was quite effective in controlling the condition.

Rhinoscleroma, or more properly scleroma, is a chronic infectious granuloma caused by the encapsulated Bacillus rhinoscleromatis or Frisch's bacillus. It occurs endemically among the inhabitants of central and southeastern Europe and sporadically in various other parts of the world, although chiefly among emigrants from these regions. A case of this type in which a native-born American was affected was reported by Thompson and me in 1928. The condition is more common among adult females and is intimately associated with a lack of hygienic surroundings. The infection usually starts in the mucous membrane of the anterior nares, the lesions appearing as edematous, bluish-red, discrete nodules which may remain localized in this region or spread onto the upper lip, the external nose, or into the palate, pharynx, larynx, or trachea. A large, ulcerated, granulomatous mass often protrudes from the nostrils and emits a foul fetid discharge so that the entire picture may strongly suggest a highly malignant neoplasm. The primary infiltration and hyperplasia is later followed by a sclerosing process and the mucous membrane becomes scarred and atrophic. A characteristic concentric

stenosis of the nasopharynx, with retraction or destruction of the uvula, is commonly present and in itself is highly suggestive of scleroma. Marked deformity of the external portion of the nose and distortion of the larynx may also occur. The disease runs an extremely chronic course; it may continue for from fifteen to thirty years and then gradually subside spontaneously. The histopathologic picture, as emphasized by Montgomery, is diagnostic and consists of: (1) the presence of Mikulicz or foam cells containing Frisch's encapsulated bacillus; (2) numerous plasma cells, and (3) so-called Russell bodies. Irradiation is the most effective form of therapy. Culture of a biopsy specimen with demonstration of Klebsiella rhinoscleromatis clinches the diagnosis.

Actinomycosis, although commonly encountered in the submaxillary, cervical, temporal, parotid regions and cheeks, occurs only infrequently in the mouth and pharynx. In the former situations, secondary extension with involvement of the ear and mastoid may take place and an abscess may rupture into the external auditory canal. In the mouth and throat, the infection is most often seen in the body and base of the tongue. The onset may be acute with severe, throbbing pain, local tenderness, general malaise and fever, as in pyogenic abscess, or insidious with slow progression during the course of several months. The latter is the rule in the absence of secondary infection. Frequently the tumor in the tongue appears on clinical examination to be a benign fibrous nodule or an infected cyst. Within the course of a few days, however, the process may flare up acutely and a large portion of the tongue may become involved. When the posterior portion of the tongue is affected, dysphagia may be so extreme that the patient is unable to swallow even fluids. If the abscess is not drained and if the nature of the condition is not recognized, spontaneous rupture with subsequent repocketing and extension of the infection occurs. Ultimately, the entire tongue and floor of the mouth may become involved and the process may extend through and present in the submaxillary and submental regions. The diagnosis is based on a demonstration of so-called sulfur bodies or actinomyces. Under intensive treatment with iodides, irradiation and drainage (or excision in cases with localized lesions), the prognosis is good depending on the situation, extent and virulence of the infection.

Blastomycosis,[3] while usually a systemic or cutaneous disease, may be localized primarily in the mucous membranes of the mouth or throat. In these situations the lesions present much the same clinical appearance as those involving the skin, that is elevated verrucous

plaques which tend to clear in the center and spread about the periphery, where minute yellowish abscesses are visible with a hand lens. Blastomyces may be demonstrated in the purulent material expressed from these abscesses. The organisms, which appear as budding yeast forms, are also demonstrable in miscroscopic sections of the involved tissues. When the lesions occur in the mouth and especially in the larynx, the clinical picture may be very suggestive of the verrucous type of tuberculosis and only careful study of a biopsy specimen will permit differentiation.

Torulosis, a rare disease due to the pathogenic yeast, Torula, which closely resembles Blastomyces, most often involves the brain and meninges. A localized form of the infection occurs in the upper respiratory tract and at times in the middle ear. This probably precedes systemic invasion by the organism. Cases in which the infection was localized in the pharynx have been reported by Jones, Gill and others. While the disease in the brain and thorax is invariably fatal, the infection in these early cases was arrested by roentgen therapy and the administration of iodides. Jones made the interesting observation that, in half of the twenty-two cases reported up to 1927, the infection was apparently primary in the nose, throat, or ears. The causative organisms, torulae, are double-contoured, refractile and budding, and similar in appearance but smaller than the blastomycetes. They may be demonstrated in smears of the discharge or in microscopic sections of the granuloma. In the tissues, the lesions of torulosis, blastomycosis and coccidioidal granuloma are very similar and definite differentiation is largely dependent on cultural studies.

Coccidioidal granuloma, which is endemic in the San Joaquin Valley in California, often strikingly simulates blastomycosis and, like the latter condition, may produce oral and pharyngeal ulcers and granulomatous lesions. It is due to the Coccidioides immitis, a double-contoured organism similar to Blastomyces and Torula, but much larger than either of them. It reproduces by sporulation, never by budding as do the last named, and it may be demonstrated in smears or microscopic sections treated with potassium hydrate solution.

Leprosy, another of the chronic infectious granulomas, is due to the lepra bacillus. In the early stages it commonly affects the external nose as a nodular infiltration which later extends to involve the nasal mucosa and the palate, pharynx and larynx. Ulceration promptly develops in the involved mucous membrane and the secre-

tion covering these lesions contains numerous clumps of lepra bacilli resembling bundles of cigars. As the active process subsides, atrophy and cicatrization with more or less distortion occur. The lesions of leprosy strongly resemble those of syphilis and tuberculosis and, as both the Wassermann and tuberculin tests are positive in cases of leprosy, diagnosis is often difficult. In addition, the lepra bacillus is, like Mycobacterium tuberculosis, acid-fast, but it is non-pathogenic to animals.

Thrush, or moniliasis, may produce cutaneous lesions, but it much more commonly involves the oral mucous membrane, either as an acute or a chronic process. The causative organism is the parasitic fungus, Monilia albicans. The acute form of the disease occurs most commonly among suckling infants and malnourished children less than 5 years of age. It is also seen among aged or debilitated individuals, especially during the later stages of tuberculosis and other wasting diseases and during convalescence following severe abdominal surgical operations. The condition is characterized by whitish or grayish, curdy exudative plaques covering a varying amount of the oral mucosa. As a rule this can be wiped away readily and leaves an extremely tender, excoriated and often oozing surface. The intense discomfort often associated with the disease may interfere markedly with the patient's nourishment. The clinical findings are characteristic and the diagnosis is confirmed by demonstration of yeast forms and mycelium in films or cultures of the membrane. The condition can often be cleared up with a single, thorough, local application of 10 per cent formalin following cocainization.

So-called submucous or chronic thrush is a more deeply seated process than the acute form. It occurs almost exclusively in adults, many of whom are in good general health otherwise. It is characterized by the presence of whitish, thread-like lesions within or below the mucous membrane. The entire surface of the tongue, as well as most of the remainder of the oral mucous membrane, may be infiltrated by the process, and this same involvement may extend through a varying portion of the gastro-intestinal tract. When the process is well localized in the mouth, destruction of the areas with the actual cautery or with a fine diathermy electrode, followed by the application of a 2 per cent solution of gentian violet is quite effective in relieving the discomfort, although complete eradication of the process is uncertain. The exudative type of chronic thrush infection is at times extremely persistent and has in some instances

appeared to be a definite etiologic factor in the development of carcinoma of the buccal cavity.

In the differential diagnosis of submucous thrush, linear leukoplakia, lupus erythematosus and lichen planus must be ruled out. Considerable difficulty is often experienced in excluding the last named condition, although the cutaneous lesions of lichen planus, which are frequently situated on the wrists and other flexor surfaces, are of help. The oral lesions of the disease may occur in any portion of the mouth and they commonly appear as grayish striæ which often form a diffuse network and at times thickened leukoplakia-like plaques. Histopathologic studies frequently are necessary for differentiation. According to Montgomery, these reveal a more edematous and boggy epidermis in submucous thrush, also definite spores and mycelial filaments in the tissues. Lupus erythematosus of the lips often produces a peculiar "silvering" due to adherent white scaling.

A case of primary tularemic ulceration of the pharynx was reported for the first time by McGovern in 1936. This condition is extremely rare, but it must be borne in mind as a diagnostic possibility when patients present extensive ulceration of the throat. In McGovern's case there were large confluent ulcerations of the pharynx and the cervical lymph nodes were enlarged and tender. The onset of the illness had been acute, with chills, severe sore throat and fever up to 104 degrees F. The diagnosis of tularemia was based on strongly positive agglutination tests. A 1 per cent mercurochrome throat spray gave prompt and marked relief and the patient recovered without complications.

The earliest manifestations of pemphigus are not infrequently encountered in the mouth, pharynx and larynx; rarely are they encountered in the nose. During the course of the disease the eyes are commonly involved and, at times, the ears as well. The characteristic primary lesions, bullæ on a noninflammatory base, may appear in the mouth or throat some little time prior to their development on the external body surface. They are usually only a few millimeters in diameter, but may range up to 1 cm. or more. They are extremely thin-walled and inconspicuous and usually rupture within the course of a few hours. Careful observation of the patient for several days frequently is necessary in order that these bullæ may be detected. Following rupture the flaccid pellicle sloughs off, leaving a superficial painful ulcer which promptly develops a marked secondary inflammatory reaction. These ulcers heal slowly and, as they often involve a considerable portion of the mucous membrane

of the mouth and throat, the patients frequently are extremely uncomfortable. Treatment has not proved effective in controlling the condition to date and in the severe cases the prognosis is fatal.

Bullous lesions in the mouth may also be produced by erythema multiforme. However, this condition is as a rule readily differentiated by the history of similar attacks, which disappeared spontaneously, the seasonal incidence in the fall and spring, and by the multiformity and distribution of the lesions, especially on the extremities. Various drugs may likewise cause lesions in the mouth that might be confused with erythema multiforme, among them phenolphthalein, antipyrine, acetanilid, quinine, and the bromides, iodides, barbiturates and others.

A group of cases in which there was permanent enlargement of the lips and face (reported from The Mayo Clinic by New and Kirch a few years ago) are of interest in this connection. The condition occurs most commonly in young adults and is preceded by periodic attacks of swelling which most frequently involve the lips and cheeks, but may affect the nose, eyelids, tongue and soft palate. The swelling develops in the course of a few hours, often without premonitory symptoms, and requires two weeks or more to clear up. After a second or third episode the swelling persists between the attacks so that the affected parts remain diffusely enlarged and the tissues firm and inelastic. Frequently the acute attacks are accompanied by facial paralysis on one or both sides. This often clears during the interim between attacks. The permanent enlargement does not progress beyond a moderate degree, although it is often sufficient to produce grotesque disfigurement. It is always less than the size attained during the acute stage. The condition is not preceded by lymphangitis or erysipelas. Histologically, there is edema and lymphocytic infiltration only. The process has been controlled with injections of hot water or alcohol into the subcutaneous tissues and by external irradiation with subsequent plastic surgical procedures to shape up the parts if necessary.

Allergic reactions to a great variety of foods, certain cosmetics and clothing may be responsible for dermatitis of the external ear and for gingivitis and stomatitis in sensitive individuals. The offending irritant in such cases frequently can only be determined by careful observation and by sensitization studies.

Aspergillosis[9] due to a group of pathogenic molds most commonly occurs in the external auditory canal and in old pulmonary

abscess cavities. Varieties of the Aspergillus usually encountered are the niger, flavus and fumigatus. Of these, the last is the most frequent invader. The presence of cerumen favors the development of the fungus in the ear canal. Clinically the condition appears as a dark moldy membrane which may produce only blocking of the canal with mycelium and resultant impairment of hearing, or there may be ulceration and suppuration of the wall of the canal or penetration of the membrana tympani and involvement of the middle ear. Rarely, the mycosis involves the mouth, nasal fossa, antrum, ethmoids, sphenoid sinus and orbit. Treatment consists in improving local hygienic conditions, together with antiseptic applications, such as 50 per cent alcohol containing small amounts of bichloride of mercury and boric acid or mercuric iodide.

Furunculosis, herpes, eczema, seborrhea and erysipelas of the ear are familiar to all otolaryngologists and need only be mentioned.

Dissimilar[10] metallic dentures in the mouth at times cause local irritation and even leukoplakia in some cases by virtue of galvanic phenomena. In my experience, however, oral lesions due to this type of reaction are rare.

Aphthous and herpetic stomatitis are also familiar to all and need not be discussed.

Leukoplakia may occur in the mouth, pharynx, or larynx, but is most frequent in the buccal cavity. It is evidence of a defensive reaction on the part of nature in an attempt to protect the oral tissues from some form of chronic irritation. Tobacco is the most common irritant responsible for it, but dental factors, habitual biting of the lips and cheeks, and hot and spicy foods may play a part. Several distinct types of leukoplakia occur, and their appearance and clinical behavior vary considerably. Among these may be mentioned: (1) the squamous type, which is the most common form; (2) the papillary type; (3) that in which atrophic syphilitic glossitis is the predisposing cause, and (4) that in which interstitial syphilitic glossitis is present. The squamous type may or may not be associated with syphillis. It is of no consequence unless it becomes thickened and leathery, in which event its removal by electrocoagulation or the actual cautery is indicated because of the danger of malignant change. Prior to the development of thickening, removal of the cause will usually be followed by spontaneous clearing of the condition. Papillary leukoplakia is a potentially malignant lesion, tending to develop into the so-called frozen-doormat type of low-grade squamous-cell epithelioma. It appears as a thick, tough, warty,

insensitive area and, like other forms of leukoplakia, is often discovered accidentally by the patient. The form of leukoplakia developing on the basis of atrophic syphilitic glossitis is also very prone to undergo malignant change and areas of ulceration or thickening arising in it must be viewed with suspicion and removed promptly. The tongue in such cases presents a smooth, glistening, ironed-out, atrophic appearance and traumatizes readily. Carcinoma developing on this basis is usually of the active, squamous-cell type. When interstitial glossitis is present, secondary lobulation of the thickened grayish tongue surface results.

Keratosis pharyngis is characterized by the presence of numerous grayish, whitish, or yellowish horny out-growths which appear most commonly on the faucial tonsils, but not infrequently on the lingual tonsils and, at times, on the nasopharyngeal adenoid pad and lymphoid tissue elsewhere about the pharynx. This condition, which is caused by Leptothrix buccalis, is often symptomless and frequently is discovered accidentally. When forcibly removed, the adherent horny protuberances leave a denuded, bleeding surface and tend to reform promptly. They are prone to disappear spontaneously in the course of time and treatment is usually not indicated. If the condition is causing appreciable pharyngeal irritation, electrocogulation of the individual points is justifiable.

Black tongue[11] or hairy tongue appears primarily as a black or brownish hairy patch on the posterior portion of the dorsum of this structure just anterior to the circumvallate papillæ, but the condition may involve the entire dorsum. Histologically, the long hair-like processes on the surface of the tongue originate from abnormal papillæ in the epithelial covering. According to Andrews, the color is due to changes in the horny cells analogous to those seen in certain types of hyperkeratosis of the cutaneous surface or to pigment from substances taken into the mouth. A foul taste is at times complained of by the patient but, more often, there are no associated symptoms. Treatment is usually of little avail. The condition often clears spontaneously, but it may recur.

Another form of pseudoblack tongue is caused by a fungus which grows as a mold on the dorsal surface.

A degree of pigmentation of the buccal surface and alveolar processes is at times seen normally. As a rule, however, pigmentation of the mucous membrane of the nose, mouth and throat is pathologic. It may occur in various systemic diseases, such as hemochromatosis, Addison's disease, and acanthosis nigricans. It may also

follow treatment with some of the heavy metals, namely, silver, gold, lead, bismuth and mercury, and also accidental or occupational poisoning by some of these, or phosphorus and similar chemicals. The blue line of lead and bismuth intoxication extending along the gingival border is well known. Extensive, diffuse, deep pigmentation of the mucous membrane of the nose, mouth and pharynx is at times seen extending widely about the periphery of melanotic epitheliomas in these situations.

Hypertrophy of the lingual papillæ, producing furrowed or so-called scrotal tongue, geographic tongue, burning tongue and increased accumulation of lymphoid tissue along either lateral lingual border posteriorly, often cause much concern to the patient as well as to the physician who is unfamiliar with these conditions. None of these conditions is of clinical importance, however, and no local treatment is required. Ample reassurance is essential as fear of malignancy often forms the basis for complaints in this connection. Burning tongue as a rule is due to a psychic disturbance.

Glossitis rhomboidea mediana designates a benign lesion of the tongue, often slightly elevated and of an ovoid or rhomboid shape, situated in the midline of the dorsum just anterior to the circumvallate papillæ. The condition is usually symptomless and in most instances is discovered accidentally. It is frequently mistaken for carcinoma.

So-called Fordyce's disease is the anomalous presence of numerous enlarged sebaceous glands in the oral mucosa. These appear as minute to pinhead-sized whitish or yellowish granular nodules within the mucous membrane, most often on the posterior portion of the cheeks and in the lips near the angles of the mouth. The condition is not pathologic and no treatment is indicated.

Benign hypertrophy of the lymphoid tissue over the base of the tongue and along the lateral pharyngeal folds is at times pronounced and necessitates treatment. Electrocoagulation, with or without direct application of radium to the surface, and external irradiation, followed by the administration of iodides, is the most effective treatment.

Xanthomas of the pharynx and larynx are rare. Two cases were reported from The Mayo Clinic by Finney, Montgomery and New in 1932, and New subsequently reported two additional cases (1935). The etiology of xanthoma is not known, but an abnormality of lipid metabolism associated with some local condition is thought to be responsible. In some cases the condition is associated with diabetes

mellitus. Multiple yellowish nodules or plaques of varying size and distribution may be present in the mouth, pharynx, or trachea. These may be of sufficient size and extent to produce marked hoarseness. The histopathologic picture is similar for all types of xanthoma. According to the foregoing authors, the process may be divided rather arbitrarily into an early or inflammatory stage, a tumor stage, and a stage of regression or fibrosis. The most prominent feature is the presence of the xanthoma or foam cells which with fat stains are seen to be laden with various lipids and which are apparently derived from the reticulo-endothelial system. "Touton" giant cells, characterized by a central circular arrangement of their nuclei and a peripheral zone of lipid-containing cytoplasm, are frequently seen, especially in the tumor stage. Treatment, aside from surgical removal of such tumors, is of little avail.

SUMMARY

Different dermatologic conditions affecting the ears, nose, mouth and throat have been reviewed. A group of these represent a purely local condition; more of them, however, are a manifestation of some systemic disease. They are of interest to the otolaryngologist not only because of the therapeutic indications arising in connection with the local pathologic changes, but also because of their diagnostic and prognostic features in general disease. Recognition is at times possible from observation of the lesions of the mucous membrane alone, but careful study of the cutaneous surface, various laboratory investigations and complete physical examination may also be required.

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IX

OSTEOMYELITIS OF THE SPHENOID BONE—REPORT OF FIVE CASES WITH THE AUTOPSY FINDINGS

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Osteomyelitis of the sphenoid bone is not common as judged from the reports in the literature. Teed[1] in 1938, after a careful search, found 129 cases of meningitis secondary to infections of the sphenoid sinus. He found in a study of the statistical reports of the literature that the sphenoid sinus was involved in about 15 per cent of clinical cases of sinusitis and in about 33 per cent of pathologic cases, but was responsible for about 35 per cent of all intracranial complications of nasal origin. The spread of the infection to the meninges was found to be predominantly vascular, particularly by way of the infected sphenoid mucosa through the vascular marrow spaces of the sphenoid to the meninges. These cases must of necessity have had some form of an osteomyelitic lesion of the sphenoid bone in all or at least in the vast majority of instances. Macroscopic disease of the sphenoid bone was observed in 52 of the 129 cases and microscopic evidence of involvement of the bone was found in nine instances in which the latter method was used.

The etiologic factor in most instances would seem to be a preceding sphenoiditis, although the possibility of a retrograde thrombophlebitis from the cavernous sinus or an extension from a petrositis should be borne in mind. An additional cause as demonstrated in Case No. 3 could be a preceding osteomyelitic lesion involving the superior maxilla, orbit and ethmoid. In rare instances an isolated thrombi or bacteria from a distant focus may conceivably lodge in the vascular spaces of the sphenoid bone as in other bony structures resulting in an osteomyelitis.

The extension of an infection from the sphenoid mucosa to the bony structure or to the meninges occurs in most instances as pointed out by Turner and Reynolds[2], and by Eagleton[3] by a suppurative thrombophlebitis of the small vessels.

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Teed failed to find evidence of transmission, which reached the meninges, by way of a dehiscence.

The possibility of the sphenoid bone becoming infected from an involved cavernous sinus was demonstrated by Turner and Reynolds[2] in the report of a case in 1928. The relationship of the cavernous sinus and the sphenoid is very close, as only a very thin bone separates them. Numerous anastomosing veins are valveless (Campbell[1]) so that transmission may be in either direction.

As the tip of the petrous portion of the temporal bone is in close proximity to the sphenoid bone, infections of the tip may extend to the sphenoid. Eagleton[3] called attention to this in 1932. Since that time several cases have been reported. This method of transmission is illustrated in Case No. 1.

The diagnosis of an osteomyelitis of the sphenoid bone before the advent of intracranial complications is difficult. The symptoms of a sphenoiditis would be present in most cases. These may be vague, such as those produced by any subacute postnasal inflammation. There may be a deep seated headache or a headache behind the eyes, or it may radiate to the temporal or occipital regions. Eye symptoms such as photophobia, lacrimation, scotomas, or blepharospasm may be noticed. A postnasal discharge is usually present.

As invasion of the body of the sphenoid bone occurs, symptoms and signs of sepsis may ensue, although localizing symptoms may be absent. The septic symptoms before intracranial involvement has occurred are usually mild; the temperature is frequently low and the toxemia may not be marked. Kramer and Som[5] emphasize the temporal or retro-orbital pain especially marked at night. This latter symptom is equally characteristic of a petrositis. The intranasal examination usually reveals nothing other than the usual signs of a sphenoiditis.

A roentgenologic examination may demonstrate a cloudy or vague outline of the sphenoid sinus wall. This increased density or cloudiness may extend into the middle fossa or along the base of the skull and is especially marked on the side of involvement. If the infection is bilateral then both sides would be equally cloudy. The sella turcica may show changes associated with an osteitis or an osteomyelitis. According to Pfahler[6] the sella turcica may show calcification of the bridge or of the ligaments connecting the anterior and posterior clinoid processes. The bands posterior to the posterior clinoid process may also show a calcification or a flocculent lime deposit according to Pfahler.

The organisms usually responsible for the osteomyelitis are the pneumococcus type III and the hemolytic streptococcus. Other pyogenic organisms are occasionally recovered. Four of the five cases reported here were due to the pneumococcus type III. In the fifth case a hemolytic streptococcus was found.

The most frequent complication of an osteomyelitis of the sphenoid is a meningitis. The next most frequent complication is a thrombosis of the cavernous sinus. Teed[1] found the infection extended to the soft tissues at the base of the skull with extension to the face or neck in seven of the 129 reported cases. Meningitis was present in five of these cases. The pituitary gland is occasionally involved with a possible production of a glycosuria.

The following cases, proven by autopsy, have been seen at the Evanston Hospital between the years 1923 and 1938. A number of cases of death from meningitis in which a clinical diagnosis of osteomyelitis of the sphenoid bone was made or could have been made was found. These were excluded because of lack of autopsy or incomplete autopsy; that is, microscopic sections were not made of the sphenoid where macroscopic evidence of disease was absent.

REPORT OF CASES

CASE 1.—J. G., a boy five years of age, entered the Evanston Hospital on April 12, 1923. Four weeks previous to his entrance he had had a sore throat with an earache in the left ear. The ear had a slight discharge which was said to have stopped after five days. One week before entering the hospital he complained of feeling tired and sleepy, followed by abdominal pain, stiffness of the legs and neck, and a temperature varying from 101 to 106 degrees F. He gave a history of having had two previous attacks of acute suppurative otitis media which had healed in a satisfactory manner.

The examination on entrance revealed a mentally alert child, but evidently quite ill. The left pupil was larger than the right. Both ears were negative on external examination with the exception of some slight tenderness on pressure over both mastoids. Examination of the tympanic membranes revealed an intact drum on the right side and a small perforation in the upper anterior quadrant of the left membrane. Spinal puncture taken at nine o'clock on the morning of entrance revealed a clear fluid containing ten cells (polymorphonuclear leucocytes). The same evening another spinal puncture revealed a turbid spinal fluid with many hemolytic streptococci. The signs and symptoms of meningitis rapidly ensued, with death on the sixth day.

Autopsy: Autopsy revealed in the right middle ear cavity and mastoid cells a thin, slightly yellow, purulent material extending forward through the cancellous bone to the sphenoid as far as the anterior clinoid process. In the left middle ear cavity and mastoid there was a much larger amount of yellow, purulent material, but without evidence of osteomyelitis. The outer part of the petrous portion of the left temporal bone showed necrosis with only a thin shell of intact bone sepa-

rating the necrotic portion from the dura in the region of the superior petrosal sinus. Septic thrombi of the left lateral and left superior petrosal sinuses were found. The sphenoid and ethmoid sinuses were filled with greenish-yellow purulent material. The left frontal sinus was small but filled with yellow exudate. The frontal sinus on the right side was absent.

Bacteriologic Examination: Hemolytic streptococci were recovered from the spinal fluid and both middle ear cavities, both by cultures and smears. In addition, cultures revealed staphylococci from both middle ear cavities. Aerobic cultures of both middle ears revealed gram negative bacilli, gram positive cocci, pointed and in pairs.

Comment: The osteomyelitis of the sphenoid was evidently secondary to the osteomyelitic process of the right ear. The left middle ear, which was draining, did not seem to be a factor in the extension of the sphenoid, although a petrositis was present. The meningitis appeared to have its origin from the ear rather than from the sphenoid.

CASE 2.—A. R., a male 47 years of age, entered the hospital on March 30, 1928. One week before admission he developed a cold and sore throat which was followed by an earache. The ear was incised two days before entrance but without a profuse discharge. The day of admission he developed severe headache and a temperature of 103 to 104 degrees F., with symptoms and signs of meningitis. Pneumococci type III were isolated from the spinal fluid and middle ear. He died thirty hours after entrance.

Autopsy: Autopsy revealed acute fibrino-purulent leptomeningitis (pneumococcus type III); acute suppurative otitis media and mastoiditis on the right side and acute sphenoiditis. The roof of the right sphenoid sinus showed osteomyelitic changes with perforation to the dura. The ethmoid cells contained a small amount of sanguinopurulent material. The left middle ear and mastoid and both right and left petrous bones were normal. The venous sinuses were normal. Cultures and smears of the spinal fluid, sphenoid sinus, right middle ear and blood revealed pneumococci type III.

Comment: In this case there was a definite extension of the infection from the sphenoid to the meninges. The middle ear and mastoid infection seemed to be coincidental, although prior to the autopsy it was assumed the meningitis was of otitic origin.

CASE 3.—G. L., a male infant three months of age, entered the Evanston Hospital September 4, 1929. Two days before entrance the baby had an acute upper respiratory infection which was followed by swelling of the inner portion of the right eyelid. A beginning exophthalmos was noted. A bloody, mucopurulent nasal discharge was present from the right nostril. Cultures from the nose (ethmoid region) taken at this time revealed a streptococcus, although later a pneumococcus type III was obtained. The day after admission the child was taken to the operating room and the right orbit and ethmoid sinus were drained through an orbital incision at the inner canthus of the eye. An orbital abscess was found which drained freely. The following day a fistula developed in the roof of the mouth

and in the canine fossa of the right side. These fistulas communicated with the maxillary sinus and the body of the maxillary bone. At this time a slight retraction of the neck, without rigidity, developed. The child seemed to improve for about one week, when a lower lobar pneumonia developed, followed by death eight days after entrance. The temperature ranged from 102 to 103 degrees F.

Autopsy: Autopsy revealed an acute purulent ethmoiditis and a bilateral panophthalmitis (orbital abscess). The orbital tissues on both sides were distended, rather tense with a thick greenish pus escaping from the left orbital cavity. The right orbital cavity was free of pus, as it had drained freely through the incision and fistulas. The sphenoid bone showed an osteomyelitis with free pus surrounding the cavernous sinus, but without a cavernous sinus thrombosis. The meninges were free from exudate. The ears and venous sinuses other than the cavernous were normal.

Cultures and smears from the sphenoid and ethmoid sinuses and from the orbits taken at autopsy revealed a pneumococci type III (gram positive cocci in pairs and clusters and diplococci in pairs and short chains).

Comment: This infant developed an osteomyelitis of the superior maxilla and of the sphenoid bone with bilateral orbital abscesses with an extension of the sphenoid infection to the outer region of the cavernous sinus. It is probable that a later development of a cavernous sinus thrombosis and meningitis would have occurred except that death from sepsis and pneumonia took place before the advent of these intracranial complications could ensue.

CASE 4.—C. B., a male 21 years of age, entered the Evanston Hospital under the care of Dr. E. M. Hartlett (through whose courtesy I am reporting this case) on October 5, 1933. He gave a history of having had a running ear (left) since ten years of age. One week before admission he developed a sore throat with fever. Two days later he developed an earache on the left side which was followed by a discharge.

On admission he had a temperature of 104 degrees F., stiffness, rigidity, and pain in the neck. Examination revealed tenderness over the left mastoid with a profuse mucopurulent ear discharge which had a bad odor. He had a white count of 37,000. He later had several projectile emeses which were thought to be due to meningeal irritation. A mastoid operation was done in which several mastoid cells were found to contain an exudate similar to that in the middle ear. The mastoid process as a whole was markedly eburnated. The dura and lateral sinus seemed to be normal. The tympanic membrane was incised following the operation. A spinal puncture at this time revealed increased pressure and a slightly turbid fluid with a cell count of one. Cultures from the left ear showed frequent colonies of hemolytic streptococci and occasional colonies of pneumococci. Cultures from the mastoid were negative. Two days after the mastoid operation he developed signs and symptoms of pneumonia and sepsis from which he expired.

Autopsy: Autopsy revealed clear cerebrospinal fluid. When the dura of the left temporal fossa was stripped from the base of the skull an oval perforation (operated) was found in the posterior aspect of the petrous portion of the left temporal bone. This led into the operated mastoid cavity. However, gross evi-

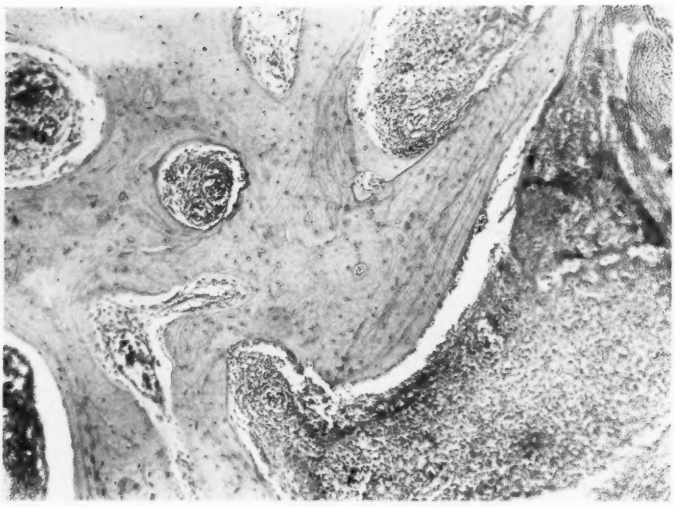

Fig. 1. Osteomyelitic process invading the marrow spaces of the sphenoid base. (Case No. 4.)

dence of suppurative inflammation other than the eburnated mastoid was not found in this region. The right middle ear and mastoid were normal.

Sections of the petrous portion of the temporal bone, including portions of the wall of the attic, showed little evidence of erosion. However, some cellular infiltration was present, probably secondary to the surgical attack in this region.

The sphenoid and ethmoid sinuses contained swollen and edematous membranes with localized areas of dense cellular infiltration. Sections of the body of the sphenoid bone (Fig. 1) revealed several areas in which the normal marrow had been replaced by dense polymorphonuclear leucocytic infiltrations. A small abscess was found within the dura overlying the sphenoid bone.

Postmortem cultures from the sphenoid and ethmoid sinuses revealed the pneumococcus type III.

Comment: This young man had a chronic suppurative otitis media, chronic ethmoiditis, subacute sphenoiditis (pneumococcus type III), and osteomyelitis of the body of the sphenoid bone with some slight cellular infiltration of the petrous portion of the temporal bone. A focal, purulent pachymeningitis over the surface of the sphenoid bone was found, indicating a localized extension to the meninges by way of the sphenoid. It was assumed prior to the autopsy that a localized meningitis of otitic origin was present.

CASE 5.—A. W. C., a male 72 years of age, entered the hospital March 4, 1933, under the care of Dr. R. H. Henderson (through whose courtesy I am reporting this case). The patient gave a history of having had "sinus trouble" for the past seven years, but no history of a discharging ear until the day of entry. There had been a progressive deafness for the past year. Two days before entrance he developed an earache. The day before admission he complained of dizziness and later severe nausea, vomiting, vertigo, associated with restlessness and nervousness. Incision of the right ear drum was done on the day of admission and thick pus obtained. Marked nystagmus was present at this time.

Examination revealed an elderly man in a stuporous condition with Cheyne-Stokes type of breathing and a discharging right ear. The left eye showed shrinking and loss of luster. The neck was stiff. The reflexes showed absent abdominals and cremasterics and a suggestive Babinski and positive Kernig on the right. Caloric tests showed normal reaction in the left ear and absence of reaction in the right ear. The urine was negative; white blood cell count was 18,000; spinal fluid, cloudy; globulin three plus, cells 4,750, predominantly polymorphonuclears. Spinal fluid culture revealed a pneumococcus type III.

On the day of admission a radical mastoid operation was done, with the removal of the bone over the roof of the antrum and middle ear. The lateral sinus was exposed but appeared normal. The symptoms and signs of meningitis rapidly ensued and the patient died 23 hours after entry.

Autopsy: Autopsy revealed a greenish-yellow exudate on the surface of the brain which infiltrated the arachnoid over the convexity; it was most abundant over the temporal and frontal regions and along the Sylvian fissures. On the inferior surface of the brain the exudate was less abundant except on the anterior surface of the pons and the lateral surface of the cerebellar lobes.

The sphenoid sinus was filled with turbid exudate containing abundant pale yellow mucopus. The membrane lining the sinus was hyperemic and somewhat thickened. It showed brownish-yellow discoloration. The nasal mucosa and the lining of the ethmoid air cells showed a similar hyperemia with a slight discoloration. The ethmoid cells contained a faintly turbid viscid mucus.

Microscopic sections of the body of the sphenoid bone showed active hematopoiesis in the marrow. The membrane lining the sphenoid sinus was thick, edematous and infiltrated with leucocytes. Microscopic sections of the hypophysis revealed polymorphonuclear infiltration of its capsule and of a very shallow zone along the surface of its parenchyma anteriorly. The pars glandularis had a normal cellular structure.

Comment: The concomitant presence of purulent ethmoiditis, sphenoiditis, osteomyelitis of the sphenoid bone and bilateral otitis media with a subacute pachymeningitis on the anterior surface of the petrous portion of the right temporal bone would seem to indicate the intracranial extension by way of the petrous rather than by way of the sphenoid.

SUMMARY

Five cases of osteomyelitis of the sphenoid bone are presented. In four the pneumococcus type III was recovered and in one a hemo-

lytic streptococcus. Extension of the infection to the meninges occurred by way of the sphenoid in two cases, and extension to the cavernous sinus in one. In two cases the extension to the meninges seemed to be by way of the petrous.

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X

DOES SUBEPITHELIAL EXTENSION OF INFECTION FROM THE UPPER RESPIRATORY TRACT TO THE LOWER OCCUR: AN EXPERIMENTAL STUDY*

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Although infections of the upper respiratory tract have long been considered causally important to infections of the lower respiratory tract, the downward routes of infection have not been entirely understood. Graham,[1] in reporting the appearance of acute inflammatory changes in bronchial fistulæ promptly following an acute upper respiratory infection, stated, "The question of how infection spreads from the upper to lower respiratory tract cannot yet be answered." There is abundant literature on such subjects as bronchiectasis, lung infections, chest conditions, pulmonary disease, and sinusitis. Hodge[2, 3] has recently reviewed the literature relating to bronchiectasis and points to the relative high incidence of sinusitis in reported cases. Ebbs[4] found at autopsy that 80 per cent of two hundred children dying of pneumonia had pus in their upper respiratory tracts. Most laryngologists now regard chronic nasal sinusitis as one of the frequent causes of chronic laryngo-tracheitis,[5] yet there has been no unanimity concerning the pathways of downward extension of the infection from the sinuses, either in acute or chronic conditions.

The aspiration route had long been assumed, and probably on a logical basis. After the development of radiopaque oils, Quinn and Meyer[5] demonstrated the presence of iodized oil in the bronchial tree after instillation into the nostrils of sleeping patients. Previously Blake and Cecil[6] introduced pneumococci directly into the lumen of the trachea in animals and found that pneumonic changes extended peripherally after the infection entered the lymphatics in the hilar region. More recently the literature has contained numerous references to lipoid pneumonitis proven to have resulted from aspiration of mineral oil introduced into the upper respiratory tract.[7, 8, 9] Thus

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Fig. 1. Antral lining from rabbit 1. x270. Frank pus is present within the lumen of the sinus. Submucosal layer is considerably thickened and infiltrated with inflammatory cells. There are numerous eosinophiles in the submucosal area; infection in this rabbit was produced by the help of previous sensitization to egg albumen.

there is adequate scientific proof to consider the aspiration route or tracheal route as an important pathway of infection from the upper to the lower respiratory tract.

Further experimental study shows the importance of other routes. The lymphatic drainage from the paranasal sinuses has been investigated by Mullin and Ryder,[10, 11] McMahon[12] and others. Recently Larsell and Fenton have shown that the combined lymphatic and hematogenous route from the sinus lining to the lung is very important.[13, 14]

There remains also the possibility of the subepithelial extension of infection from the sinus downward along the air passages. Subepithelial extension of infection from the nasopharynx and sinuses to the middle ear has been considered important by Dean.[15] It seemed desirable to determine if such a route was of clinical importance to the lower respiratory tract. Therefore, it was decided to investigate this possibility with experimentally produced sinusitis in animals. Be-

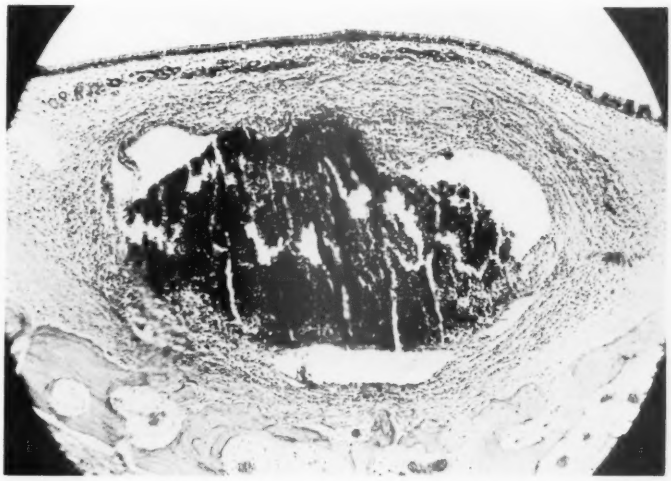

Fig. 2. Abscess in antral lining of rabbit 103. x115. Infection in this rabbit was produced by introduction of bacterial suspension after injection of water at 50° C. into the maxillary sinuses. Eosinophiles were not present as in Fig. 1. This abscess has a fairly definite capsule. The infecting organisms were secured from a human case of osteomyelitis of the frontal bone.

cause of the recent reports linking some cases of bronchiectasis with an allergic background,[16, 17] it was considered permissible to superimpose sinus infection upon allergic nasal changes produced in the laboratory. This was desirable in seeking a method of producing experimental sinusitis in animals so that the element of vaccine virus might be eliminated, as the virus was found to cause a progressive pneumonitis. This pulmonary change was found to have been reported previously by McCordock and Muckenfuss.[18] Therefore, after the first group of animals in this study had been autopsied, the method of C. S. Linton[19] was replaced by the method of L. D. Linton,[20] and sinusitis was produced in animals sensitized to egg albumen. A later small group was infected after producing pathological changes in the antral lining by irrigations of hot water, or by the use of alizarin. After a considerable variation in the duration of this sinus infection, which was substantiated by cultures, autopsy, and microscopic sections, the submucosal changes were sought from the upper to the lower respiratory tract.

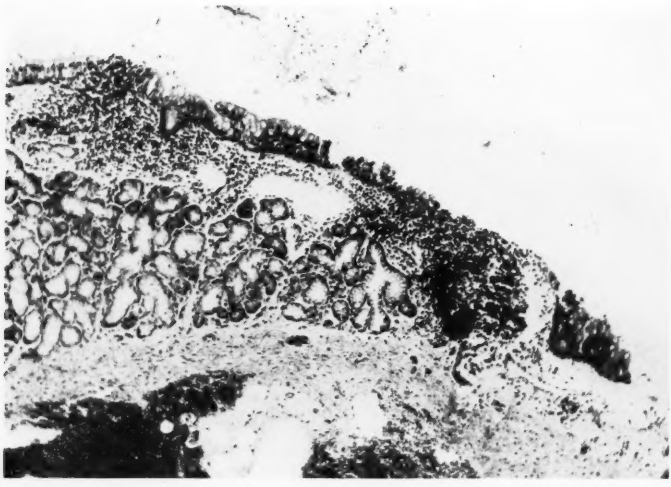

Fig. 3. Area of submucosal infiltration in nasopharynx of rabbit 100. x130. This is one of such scattered areas, and consists mainly of plasma cell infiltration and thickening of the subepithelial layer.

METHOD

Healthy, young rabbits of approximately three and one-half to four and one-half pounds were selected for this study, and of 28 injected, 20 were suitable for microscopic investigation. One died promptly of an overwhelming septicemia; another died during the night and was excluded by advanced postmortem changes; one died of anaphylaxis; others did not show adequate sinusitis.

The first group of six was sensitized by intraperitoneal, intranasal and antral injections of from .1 cc. to .5 cc. of 50 per cent egg albumen on January 30, February 6, 16, 17 and 18. On February 24, two drops of vaccine virus and .5 cc. of a heavy broth suspension of a virulent pyocyaneous culture obtained from an infected human antrum were injected into the maxillary sinuses. On the following day egg albumen was again introduced intranasally. Vaccine virus was used for this group, because up to this time we had not produced experimental sinusitis consistently without the virus and the bacterial suspension combined. These rabbits were sacrificed by a blow over the occiput from seven to thirty-two days later, and cultures and

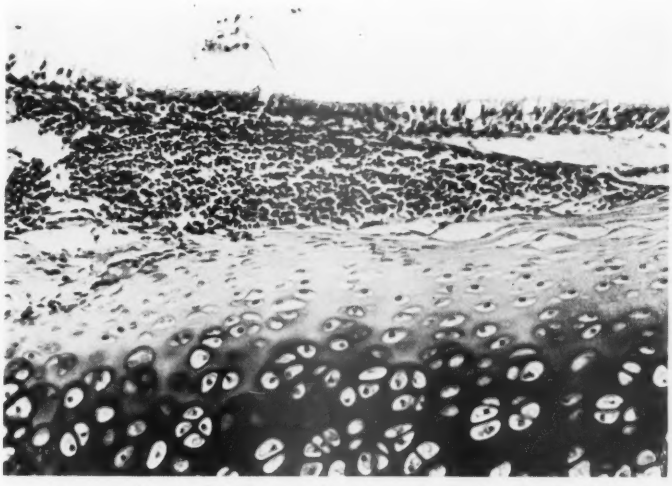

Fig. 4. Submucosal infiltration from upper level of trachea in rabbit 12. x245. This is one of several well separated areas consisting largely of collections of plasma cells in the subepithelial space.

material were obtained at immediate autopsy. The sinuses were inspected, and their lining removed for section. The lungs, trachea and esophagus, larynx and pharynx and adjacent nodes were fixed in 10 per cent formalin.

The next eighteen rabbits were similarly sensitized to egg albumen and were also infected with cultures of B. pyocyaneous or of hemolytic staphylococcus aureus without the help of virus, because of the pneumonic changes previously mentioned as being produced by the virus. Autopsies were similarly performed, and tissues were likewise removed for sectioning. The lungs were fixed by gently inflating with the formalin solution immediately so as to produce better microscopic detail.

In the last four rabbits an attempt was made to predispose the sinus lining to infection by bilateral antral irrigations with water at 50 degrees C. on two alternate days, followed by injection of virulent cultures of nonhemolytic streptococci and hemolytic staphylococcus aureus obtained from a human case of osteomyelitis of the frontal bone. There was minimal infection in No. 101, frank pus

Fig. 5. Enlarged lymph node lying between trachea, esophagus, and thyroid gland from rabbit 100. x47.

in No. 102, superficial infection and osteomyelitis at the site of injection in No. 103, and little clinical evidence of infection in the sinuses of No. 104 after an interval of three weeks. The antra of the last rabbit were irrigated with a one per cent aqueous alizarin solution, the bacterial suspension was reinjected, and clinical sinusitis developed promptly. This group was sacrificed from fourteen to forty-two days after infection. After removing the skin, the head and neck were fixed in formalin; the lungs and trachea were fixed separately. Later the larynx was dissected from the head and neck. Blocks of sinuses, nasal passages, and blocks of the nasopharynx were then cut from the head on a power saw. This last group of rabbits supplied sinus infection produced without the influence of allergy necessary to the development of the sinusitis in the other rabbits.

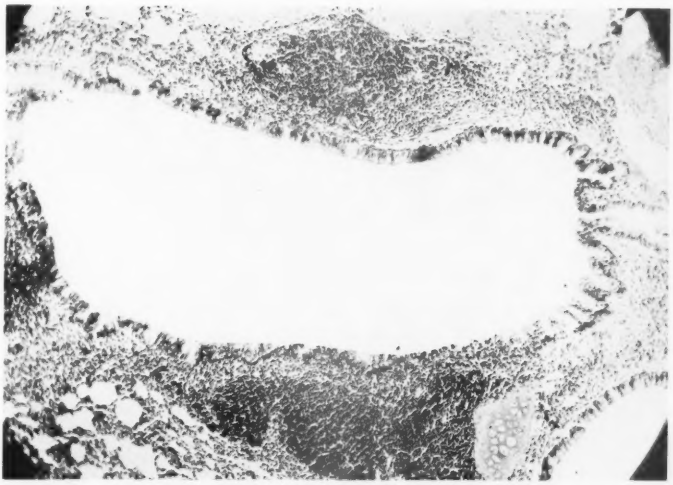

Fig. 6. Small bronchiole from rabbit 19 showing enlargement of lymphoid follicles. x92. Similar changes were prevalent in the majority of animals infected.

Routine microscopic sections were prepared, using hematoxylin and eosin stains. Tissues containing bone were decalcified in the usual manner. Sections were made to show the region from the nasopharynx to the lung periphery. In some the larynx and trachea were sectioned longitudinally; in others cross-sections were made at frequent levels. In this manner, if significant submucosal changes were produced, they should easily be found under the microscope.

Control studies were made on healthy, uninfected rabbits, and on rabbits sensitized to egg albumen but without infection. All microscopic and gross studies are as compared with the control animals.

GROSS AND MICROSCOPIC OBSERVATIONS

A variable degree of infection was present in the sinus linings. Practically all injected sinuses contained pus grossly, and the infecting organisms were recoverable by culture. Most showed thickening of the subepithelial layer and infiltration with inflammatory cells. A moderate number of eosinophiles were present in some. Several con-

tained large collections of polymorphonuclear cells within the lumen of the sinuses; a few contained abscesses within the sinus lining. Some contained minimal microscopic evidence of infection.

Careful study of many sections from the pharynx, larynx and trachea showed relatively less submucosal change than expected. There were a moderate number of scattered collections of plasma cells and areas of increased connective tissue cells here and there in the nasopharynx, pharynx, epiglottis, larynx and trachea. Some eosinophiles were occasionally seen, as would be expected with the allergic factor in the production of the sinusitis. These changes were not continuous, nor were they sufficiently consistent or of sufficient degree to indicate that the subepithelial route can be of especial importance in the downward extension of infection in rabbits.

Little change could be found in the subepithelial areas of the bronchi, but there was a very definite increase in size and numbers of the lymphoid follicles adjacent to the larger and smaller bronchi in practically all the lung sections of the infected animals as compared with the control group. Considerable interstitial fibrosis was present in the lung sections of the animals receiving vaccine virus.

Along with the increased size of the lymphoid follicles in the lung, the cervical lymph nodes were definitely enlarged over that of the controls. In the mediastinum, lymph nodes were identified from thymus tissue in the infected group, and not in the controls. Enlarged nodes were also found between the trachea or larynx, and the esophagus. The number of germinal centers in the uninfected and infected rabbits' lymph nodes was not significantly different, but the great increase in size in the infected animals was the striking finding grossly and microscopically.

SUMMARY

Experimental sinusitis was produced and proven in rabbits by postmortem findings, cultures, and by microscopic findings. Only very moderate submucosal changes were found in the respiratory tract below the sinuses. These changes mainly consisted of scattered collections of plasma cells, and some areas showed increased connective tissue elements. The most significant finding consisted of enlargement of lymphoid tissue in the neck and in the lungs. These findings are neither sufficiently definite or consistent to indicate that the subepithelial space is a very important path or route for the downward extension of infection from the upper to the lower respiratory tract, and instead lend more evidence in favor of the lymphatic route.

CONCLUSIONS

1. Experimental studies in rabbits fail to show a definite subepithelial extension of infection from the sinuses to the rest of the respiratory tract.

2. Definite changes in lymphoid tissue in rabbits favor the lymphatic route of such extension.

3. From these and other studies, it would seem that in man the combined lymphatic-hematogenous route and the tracheal route are the significant pathways of extension of infection from the upper to the lower respiratory tract.

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XI

AN OPERATION FOR POSSIBLE ALLEVIATION OF CERTAIN CASES OF CONGENITAL DEAFNESS AND CERTAIN TYPES OF ACQUIRED CHRONIC DEAFNESS*

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As is well known, deafness may be either congenital or acquired, and many forms of acquired middle ear deafness tend to be progressive. The disability of the sufferer is such that any measure that even alleviates it is one worthy of careful consideration. Besides the handicaps of deafness, some of these cases suffer from tinnitus, the relief of which to the patient would be gratifying even if the deafness were not improved.

CONGENITAL MALFORMATION OF THE AUDITORY APPARATUS

Just how frequent major congenital deformities of the auditory apparatus are is difficult to state. I have a record in a period of thirteen years of about fifty. Minot and Talbot and others have shown that arrest or acceleration of development of one or more of the six tubercles which form the ear is the basis of the various congenital deformities.

The point we wish to emphasize herein is that, although in a major auricular deformity of the arrested type, the external auditory canal, as a rule, is obliterated, and although often the middle ear is involved and may be functionless, in certain of these cases the middle ear does function. In such a case hearing may be improved by reconstructing the external auditory canal. In two cases of congenital atresia of the external auditory canal, a partially functioning middle ear was thought to have been located after exploration by a postauricular approach and, by means of a pedicled skin flap inserted into the aditus, some improvement of hearing occurred. We have at the present time another case under observation who has pain in the middle ear indicative of a catarrhal otitis media in which operation is being considered.

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In Gould and Pyle's "Anomalies and Curiosities of Medicine" this comment is made: "A little girl under Birkett's care at Guy's Hospital more than answered MacBeth's requisition 'Had I three ears I'd hear thee!' since she possessed two superfluous ones at the sides of the neck." Early Apert and Sasspedatelow reported rudimentary ears on the neck. However, it is probable that congenital skin and cartilaginous growths along the side of the neck are not really auricles, but misplaced cartilaginous remnants from branchial arches lower than the first and second from which the auricle or true auricular appendages develop. Two auricles have been reported by Knapp and more than two by Cassebohn and Langer. In our series there is one patient who had a fair shaped auricle posterior to the meatus and a smaller one anterior to it and on the same side. As is not uncommon, in this patient the side of the head, the cheek and the mandible were underdeveloped and a unilateral macrostomia was present. Guys, the celebrated writer of the Eighteenth Century, was born with only one ear. In 1859 Stahl called attention to the fact that deformity of the auricular cartilage might be regarded as an indication of arrest of development of the skull and that it bore a relationship to the development of the skull. Bilateral absence of both ears is quite rare, but there have been three cases in our records. There is a species of sheep, native in China, called the Yougti in which this anomaly is quite constant.

Undoubtedly, complete obliteration of the auditory canal is often associated with a maldevelopment of the middle ear and sometimes also of the internal ear. Schwartze, for instance, contends that when a grave atresia is present invariably one will find the labyrinth maldeveloped. In such cases the tuning fork when placed on the vertex is as a rule heard only in the normal ear.

Rarely the tragus may be twisted so as to close the meatus. The amount of congenital atresia may vary from a partial closure to a complete obliteration of the canal. The auditory meatus may appear without a depression or with a cup-like depression or a small cul-de-sac may be present. Acuity of hearing is not materially influenced if the external auditory canal is not occluded, although the auricle may be very rudimentary. Kopetzky, however, quotes the case of a student at New York University with both ears absent, yet his hearing was fairly good.

The parents of these individuals with a major congenital deformity of the auricle ask the reconstructive surgeon two questions: First, can the ear be rebuilt so as to give it a fairly normal appear-

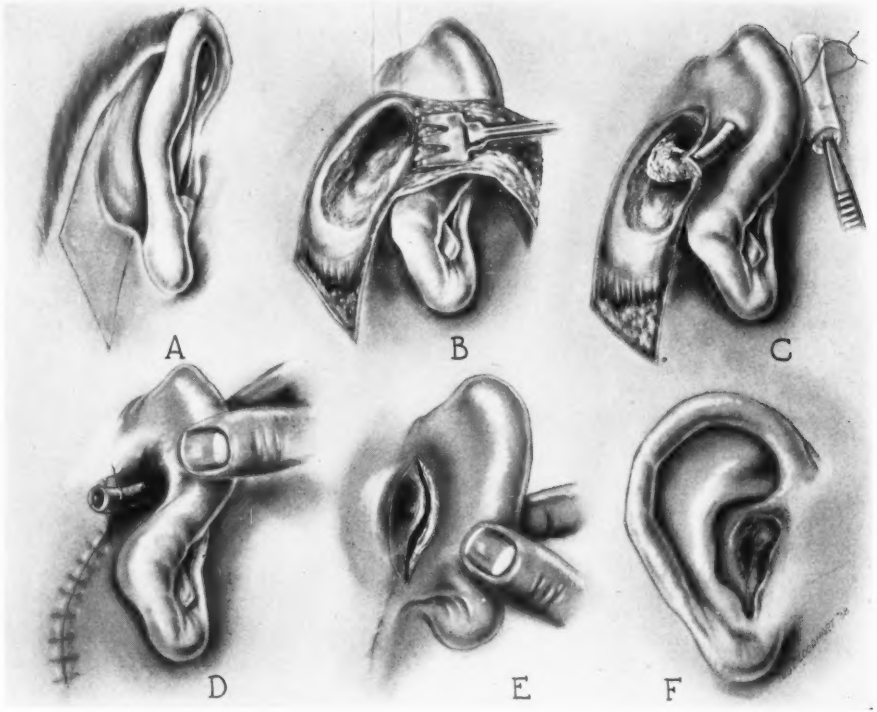
ance? (In a recent article "Total Reconstruction of the Auricle, Surgery, Gynecology and Obstetrics, 1938, 67:761-768," this question is discussed). Second, is there anything that can be done to give air conduction so that hearing will be established if the external auditory canal is obliterated?

Surgical intervention for the purpose of building an external auditory canal is not to be considered until one suspects the presence of a functioning auditory apparatus.

If there is a deformity of the auricle, especially if atresia of the meatus is present, the movement of the soft palate during phonation should be tested. When the deformity is unilateral and both arches move symmetrically it becomes a possibility that the eustachian tube and middle ear are in a more or less normal condition. On the other hand, when the opposite is found, impaired development of the tympanic cavity and eustachian tube may be assumed with probability. Malformation of the middle ear is often associated with hypodevelopment of the muscles of the palate and eustachian tube. Acupuncture may demonstrate whether the atresia is fibrous or membranous. Bony closure may be determined in this manner. A study of roentgenograms taken stereoscopically may be of considerable aid. In a child it is impossible to do a Weber test, but whenever the intelligence of age is sufficient, the Weber test will show lateralization of the sound in the malformed ear if the labyrinth on that side is functioning.

Surgical interference is to be encouraged if speech is understood on applying the ear trumpet and if during catheterization of the eustachian tube, air can be heard to enter the middle ear on auscultation. Surgical interference in bilateral deformity is demanded if there is anything in the examination to suggest even a partially functioning middle ear. When there seems to be a possibility of a functioning middle ear exploration would seem to be permissible if the parents felt that they want the utmost done for their child. Exploration can be done quickly and should have no mortality.

Although acupuncture and the roentgenogram show the atresia to be fibrous and not osseous an inter-meatal plastic operation is not effective. The middle ear is quite deep from this approach and one cannot tell with accuracy when it is entered. Even if one could enter the middle ear by an external auditory canal approach, to reline the canal with skin one is limited to either a stent graft or a pedicled skin flap. A stent graft would in all probability contract and it would seem obviously impossible to get a thin enough and


A
B
C
D
E
F

OPERATION I—FIGURES A, B, C, D

To Construct an Open Auditory Canal

A. Skin flap outlined over hairless portion of the mastoid process with pedicle toward the auricle. The flap is elevated along with the periosteum and the cortex of the bone is exposed.

B. Canal made through the mastoid process. It should be of sufficient caliber to allow the flap adequate room but it should not be so large as to leave a dead space outside of the tubed flap.

C. Pedicled flap tubed with epithelium inside about a tube. A double needled stitch transverses the auricular soft tissues as high as possible and is tied over a dental roll. This stitch is placed about one-fourth inch from the distal end of the flap. The flap should project slightly above the tube.

D. Wound closed. Stitch through tube to hold it well up in place.

OPERATION II—FIGURES E, F

To Transplant the Meatus Forward at Second Operation

E. If one wishes the external open meatus transplanted forward to the cavum concha, an incision is made completely through the auricle into the cavum.

F. The proximal end of the tube is then transplanted forward.

long enough skin flap to line the canal without obliterating the canal even if one could get the tip of the flap to the middle ear cavity. In two of these cases I attempted to enlarge the bony canal, but came to the conclusion that the procedure was not applicable. In the second case I got a temporary paralysis of the facial nerve which dampened my enthusiasm considerably. In this congenital malformation it is probable that the facial nerve is somewhat misplaced. Therefore, if after careful study, one believes the evidence is sufficient in indicating that a functioning middle ear is present and it is desirable to do something to attempt to improve the hearing, an operation for the purpose of reconstructing a new external auditory canal of the postauricular type is indicated.

THE APPLICATION OF THE OPERATION FOR CHRONIC MIDDLE EAR DEAFNESS

About a year ago Dr. O. Jason Dixon, a colleague of mine and an otolaryngologist with an inquiring mind, made the remark to me that in certain forms of chronic deafness one could open the eustachian tube, the ear or the aditus as in a mastoid operation and that while the opening remained unclosed, their hearing was much improved. The question then arose whether or not a permanent tube could be built to the aditus. I suggested that I had used such an operation to try to improve hearing in congenital atresia of the external auditory canal and I could see no reason why, in so far as the mechanics of the operation itself were concerned, the idea was not a feasible one. I then asked why he did not make a permanent opening through the ear drum. He stated that he had tried this and that the tympanic membrane always closed spontaneously.

A case of Dr. O. Jason Dixon's was selected and operated upon (April, 1938). I outlined the flap necessary to reline the bony defect to the aditus which was made through the mastoid by Dr. Dixon. I then turned the flap with skin inward and sutured it about a small rubber tube. A suture was inserted about one-fourth inch from the end of the flap and a needle placed on both ends. The needles were then placed through the soft tissues of the ear as well up and forward as possible to hold the flap entirely within the aditus. The stiffness of the tube was also used to push the end of the tubed flap well within the aditus opening by placing a stitch in it and attaching it to the skin edge after the postauricular wound was sutured together. (Figs. 1-A, B, C and D).

Later, after further consideration of the technique of this operation, it occurred to me that if one wished, at a subsequent operation

he could transplant the external opening of the new auditory canal forward to near its normal position. (Figs. 1-E, F).

After the second operation Dr. Dixon has carried on the operation in selected cases. He has now operated fifteen cases of chronic deafness. He states that in at least two-thirds of them there has been marked improvement in the hearing. In those cases suffering from annoying tinnitus, the tinnitus has disappeared which alone tended to satisfy the patient.

The exact position of the operation for the cure of deafness cannot be given at this time. Dr. Dixon soon will publish a more detailed preliminary resumé of his experiments with this operation.

Michael Jager, for certain types of deafness due to involvement particularly of the middle ear, in the latter part of the last century suggested that the mastoid be opened in a manner similar to that employed in performing the simple mastoid operation and after operation the postauricular wound be kept permanently open to permit sound waves to reach the middle ear through the opening. However, ways and means of keeping the sinus tract open were not known as they are today.

1316 PROFESSIONAL BLDG.

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XII

LATE EAR HEMORRHAGE FOLLOWING SKULL FRACTURES*

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Bleeding from an ear after an injury to the head is almost pathognomonic of a fracture of the temporal bone. In fact, with the exception of a few isolated injuries to the external auditory canal caused by a sudden impaction of the mandible against the glenoid fossa it is almost pathognomonic of a longitudinal fracture of the petrous bone. In presenting the following seven cases in which the bleeding recurred after its initial cessation, I am presenting a finding which must have been noted before, but a diligent search of the literature fails to find it recorded.

REPORT OF CASES

Case 1.—Miss A. K. was injured on April 14, 1933. While walking she was struck by a truck. Unconscious for eight to ten hours. One week after the injury she stated the right ear began to bleed. She had pain in this ear on the day following her injury. For three weeks following the first ear hemorrhage the bleeding recurred every day or every second day. The pain in the right ear, first noticed on the day after her injury, continued, but was more acute just before each succeeding hemorrhage began. She also complained of loss of hearing in her right ear which began immediately after her injury, and she stated that this deafness was more aggravated after the ear began to bleed.

When I examined her on May 10, 1933, the right drum showed no perforations. No fracture lines could made out in the right external auditory canal and no fresh or dried blood was seen in the canal.

On May 15, 1933, the bleeding from the right ear again recurred and on this date I found the right external canal filled with dried blood which was apparently coming from the superior canal wall, the drum itself being perfectly normal in appearance. X-rays at this time showed a fracture of the right temporal bone which became lost in the mastoid structure and in the petrous area. The fracture line could not be followed into the petrous bone. The examination of the hearing in this ear, made June 7, 1933, showed a marked hearing loss of the combined type.

Comment: Because the initial bleeding occurred within one week after her injury there is a possibility that she may have had a

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hematotympanum at the time of injury. No otoscopic examination was made by her physician.

CASE 2.—R. N., male, age 22, fell off a truck onto a concrete platform striking the back of his head, on December 18, 1936. There was a short period of unconsciousness. No immediate bleeding from mouth, nose or ears. X-rays taken at the hospital showed no skull fracture. At some time between four and five weeks after the accident he bled from both ears, but more so from the left ear. This was of a short duration. From then up to the time of my first examination on May 13, 1938, he states that his ears have bled off and on. He may go two weeks without any bleeding and sometimes it occurs more than once a week. The left ear bled ten days before my examination, but the right had not bled for a month. He can tell when bleeding is going to occur because his hearing gets dull and he has tinnitus just before it bleeds. After the bleeding subsides the hearing improves again. At the time of my examination stereoscopic lateral views of the skull were again taken as well as views of the pyramids in Stenvers' position, but they showed no evidence of fracture. The examination of his hearing showed a moderate perceptiion deafness in both ears.

Comment: The lack of positive x-ray evidence of fracture in this case does not rule it out. The prodromal symptoms of tinnitus and loss of hearing would indicate bleeding into the middle ear preceding the actual discharge of blood from the external auditory canal.

CASE 3.—J. Q., Male, aged 39, had two distinct head injuries in four days. On November 8, 1936, a mortar pail fell 24 feet and struck the back part of his head. On November 11, 1936, a brick fell 14 feet and struck his right occipital region. A short period of unconsciousness followed the second incident. No immediate bleeding from the mouth, nose or ears after either accident. On December 11, 1936, one month after the second injury the right ear began to bleed. On December 28, 1936, it bled again. My first examination was made on December 29, 1936. At this time no blood or clots could be seen in either ear. X-rays showed an anterior fossa fracture. The Wassermann reaction was positive. His hearing was relatively normal in both ears.

On June 9, 1937, a letter from the referring doctor stated that he himself had never seen the ear bleed but from the patient's description the discharge consisted of bright red blood. "One other doctor, who has seen the discharge described it as being rather thin and fluid but, nevertheless, the color of blood rather than blood tinged serous fluid." The doctor also stated that there were definite prodromal symptoms before the hemorrhages. These were mental depression and severe headaches usually localized in the right occipito-temporal regions. Along with these symptoms there was usually an elevation of blood pressure. At times this train of symptoms also occurred without hemorrhage from the ear.

This patient also had a spongy vascular tumor in the nasopharynx on the right side which bled easily upon manipulation.

To the best of my knowledge, the last hemorrhage from the right ear occurred on August 4, 1937.

Comment: Both injuries were to the occipital region and yet x-rays revealed a contre coup fracture of the anterior fossa. The

prodromal symptoms were evidently due to a rise of blood pressure, possibly localized in the area involved. What bearing does the presence of an angiomatous tumor in the nasopharynx have? Could there have been a similar small lesion somewhere in the ear? What role does the syphilis play in this case?

CASE 4.—Mrs. J. W., aged 36, figured in an automobile accident which rendered her unconscious for about 10 hours on June 6, 1937. Her right ear bled for two days following the accident. She had vertigo at this time and tinnitus in the right ear. On February 10, 1938, bleeding from the right ear recurred but not in large amount, but at this time the tinnitus in the right ear increased. X-rays of her skull were taken shortly after her accident and she was told that she had a skull fracture (details of this x-ray examination are not available to me).

When I first examined her on May 10, 1938, the external canal walls were negative for any fracture lines or ridges. The right drum showed no perforation, but a scar parallel to the posterior fold of Shrapnell's membrane was present. The hearing was normal in both ears.

CASE 5.—N. L. male, age 41. On may 13, 1934, a plank of wood fell a distance of 20 feet and struck him on top of the head. His right ear bled immediately and continued to bleed for two and one-half weeks. He was unconscious for four days.

About February 1, 1935, the right ear bled again during the night. No x-ray information was available.

At the time of my first examination on February 21, 1935, there was no evidence of blood in the right external canal and no signs of fracture in the canal. The right drum was retracted and scarred but showed no perforations. There was a moderate loss of hearing in the right ear, of the combined type.

CASE 6.—J. A., male, aged 36, was thrown from a truck to the pavement on August 24, 1928, and was unconscious ten to twelve hours. There was immediate bleeding from the right ear which lasted for one day. The bleeding from the right ear recurred two weeks later but lasted only a short time. X-ray examination showed a fracture extending from the right occiput across the squama and the upper portion of the external canal toward the malar. My examination was made on November 2, 1928. At this time there was dried blood on the floor of the right external canal. No fractures of the canal could be made out, but there was a scar in Shrapnell's membrane. The man complained of vertigo, tinnitus and loss of hearing in his right ear. Examination revealed a marked loss of hearing of the combined type in the right ear and a moderate loss in the left ear of the same type. There was considerable improvement in the hearing in the next four months so that there remained only a slight loss of hearing in both ears and only of a conduction type. The hearing for high tones had completely improved.

CASE 7.—H. S., male, age 41. On June 15, 1936, he fell from a ladder, a distance of fourteen feet, to a concrete driveway. He was unconscious for six days and delirious for an additional twenty-eight days and during the first seven

weeks he had no conception of his whereabouts. His wife stated that both ears bled continuously for about two and one-half months. The left ear never bled thereafter, but the bleeding from the right ear recurred every week or so up to the time of my second examination made on October 19, 1938. He complained of tinnitus in the right ear and loss of hearing in this ear. Examination showed only a few hearing rests in the right ear (Barany apparatus in the left ear). The left ear showed a slight conduction loss only. No blood was seen in the right external canal and no fractures of its walls. The right drum showed scarring in the postero-inferior portion. The original x-ray findings were negative for a skull fracture and x-rays which I had made on October 19, 1938, did not reveal any petrous fractures.

Comment: In spite of negative x-ray findings this patient sustained a bilateral longitudinal fracture of both petrous bones (long continued bleeding from both ears). The hearing of the right ear was almost entirely gone, that of the left returned almost to normal.

Bleeding from the ear is a very common symptom of basal skull fractures. Graf[1] found it in 77 out of 90 cases, Borden[2] in 221 out of 408 cases, Moody[3] in 24 per cent of his cases, Ransahoff[4] in 31 per cent, Wortis and Kennedy[5] in 34.2 per cent, Besley[6] in 31.5 per cent and Voss[7] in 32 per cent of his cases. Gurdjian[8] and Yerger[9] are of the opinion that a rupture of the drum after head trauma may be unassociated with a basal skull fracture whereas the majority of authors (Ramadier and Caussé,[10] Davis,[11] Besley,[6] Voss,[7] Phelps,[12] and W. Lange[13]) believe that bleeding from the ear after a head injury is a sure sign of temporal bone fracture. Ramadier and Caussé[10] feel that for bleeding from the ear to be symptomatic of fracture the bleeding should be immediate, abundant and persist for at least several hours. In his operative work on these cases Voss,[7] in agreement with most authors, (Barnick,[14] Linck, Lange,[13] Ulrich,[15] Brunner and Marx) could find a torn drum only when the margo-tympanicus had been reached by the fissure.

"As a rule transverse fractures of the pyramid do not involve the membrana tympani, therefore, hemorrhage from the ear in head injuries usually means a horizontal fracture of the pyramid and is almost pathognomonic of middle fossa fractures" (Mellinger[17]). This statement is only true of the pure transverse fractures and not of the combined transverse and longitudinal fractures. Even in the pure, uncomplicated labrythinthine fractures there have been a few exceptions to this rule. (Cases of Voss,[7] Biechele[18] and Schittler[19]).

The sources of the bleeding in these cases are fractures of the external canal, injury to the tympanic plexus, the sigmoid sinus, the superior petrosal sinus, the jugular bulb, the middle meningeal artery and in a few rare cases also the internal carotid artery (Mellenger[17]).

When the bleeding is especially abundant it is apt to arise from one of the peripheral sinuses. In injury to the internal carotid artery the hemorrhage is cataclysmic in character and usually occurs at the same time from ear, nose and mouth.

If the fracture line opens into the middle ear but does not reach the margo-tympanicus or the external auditory canal, the ear itself does not bleed but we find an effusion of blood filling the middle ear which causes the drum to bulge and gives to it a bluish discoloration, a condition known as hematotympanum. While bleeding from the ear is almost always evidence of a longitudinal fracture of the pyramid and practically never occurs in transverse fractures of the pyramid, hematotympanum can occur in either variety of petrous fractures.

I will now pass on to a discussion of the cases in question. All seven had a head injury by blunt force. All of these bled from the ear after the accident, four of them immediately and the other three at a variable period after the trauma. In all of the cases the bleeding recurred one or more times after a complete cessation of the initial hemorrhage. All of them had a period of unconsciousness after the injury. In four of these cases the x-ray findings were positive for some sort of a skull fracture. In Case 1 the bleeding first started one week after her injury. This might have been due to the breaking down of a hematotympanum if such a condition was present. No otoscopic examination was made at the time of injury. In Cases 2 and 3 the bleeding did not begin until four to six weeks after the accident. Interesting are the prodromal symptoms appearing in Cases 2 and 3.

That late hemorrhage from the ear after injury can occur is shown by a case reported by Forestier[20] of a child who began to have a series of hemorrhages from the external canal three weeks after the accident and this is somewhat analogous to a case reported by Vincenzo Palumbo[21] of an isolated fracture of the external canal from which an escape of cerebrospinal fluid occurred three weeks after the accident and recurred intermittently for two years before a meningitis developed. Cairns[23] has also recently reported a case of a skull fracture through the frontal sinuses which developed a cerebrospinal rhinorrhea three months after the original injury. Moreover, Urbantschitsch[22] reported a case in which seventeen days after a blow to the right parietal region a hemorrhage into the labyrinth took place evidenced by a sudden onset of tinnitus, deafness, nausea, vomiting and severe vertigo. The examination revealed complete

deafness and ablation of the vestibular function. We also know that late hemorrhages into the brain take place after these injuries.

The recurrence of these ear hemorrhages after cessation of the initial bleeding is analogous to the intermittent nose bleeds and the intermittent discharge of cerebrospinal fluid from the nose after head trauma, several of which cases I have under my observation at the present time. In these nasal cases I have also observed prodromal symptoms, similar to those exhibited by Cases 2 and 3, probably also caused by increased blood pressure and increased intracranial pressure.

Ramadier[24] is of the opinion that these recurring hemorrhages can be explained by a coincidence between the traumatism and the presence in the middle ear of a tumor such as an angioma. While such an explanation might hold in an occasional case such as Case 3, in whom a vascular, free bleeding tumor was found in the nasopharynx, it hardly seems to me to be applicable to all seven cases. Nager[25] has never seen bleeding from the ear as long after the injury as my observations indicate, but supposes that it might be caused by a longitudinal fracture by which the middle meningeal artery or one of its branches was injured and which could not repair itself because of displacement of the border lines of the fracture. This explanation does not seem to be entirely plausible for the clinical picture of middle meningeal hemorrhage is well known and does not fit any of these cases.

The only cases in which Voss[26] has seen frequently repeated ear hemorrhages have been in injuries to the lateral sinus by a longitudinal fracture. In this personal communication to me he states that Pietrantoni has also seen similar cases of repeated bleeding and has explained them as due to a rent in the sinus. In the opinion of Voss, the hemorrhage occurs through a tiny fissure in the external canal wall which is not visible microscopically. He wonders if in my cases the fluid was not cerebrospinal fluid instead of blood, and while this might possibly have been true in Case 3, it certainly was not true in the remaining six cases.

Brunner[27] suggests that these late ear hemorrhages are in a way analogous to the late hemorrhages into the brain, a condition which has been known for a long time, the explanation of which, however, is not clear (Stiefler[28]). It is also his opinion that these late hemorrhages can arise from traumatic aneurysms of small branches of the superficial temporal artery in the external canal or from like

aneurysms of small branches of the middle meningeal artery within the dura. According to him the prodromal symptoms before the bleeding in Cases 2 and 3 as well as the elevation of blood pressure noted in Case 3 could be explained by disturbances in the blood circulation occasioned by these supposed aneurysms.

The true explanation of these late ear hemorrhages will, however, probably have to remain in the field of speculation until we have the opportunity to make a thorough histological examination of the temporal bone and its adnexa in one of these cases.

WELLS BUILDING.

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XIII

PERIESOPHAGEAL ABSCESSES: THE IMPORTANCE OF EARLY SURGICAL INTERFERENCE*

WESTLEY M. HUNT, M.D.

NEW YORK

The mediastinum may become infected following a peritonsillar or retropharyngeal abscess, suppuration of the cervical lymph nodes, thryoidectomy, tracheotomy, diseases of the cervical spine, influenza bronchitis, pneumonia and pericarditis. This report concerns infection of the mediastinum in the cervical region following a rupture of the esophagus.

Ruptures following foreign bodies and malignancy must have occurred many times prior to the introduction of peroral endoscopy, thoracic surgery, and specialized x-ray technique, but the hospital records which have been examined indicate that prior to their development such mediastinal involvement may have been listed as pneumonia or cancer.

An increasing number of papers are being published by both laryngologists and surgeons reporting cases of cervical periesophageal involvement and describing a surgical approach.

The establishment of broncho-esophagological and thoracic clinics in so many city and suburban hospitals have encouraged more accurate diagnoses with resulting improvement in treatment and lessening of mortality.

Even in large centers of population one's personal experience is limited. The records of the Manhattan Eye, Ear and Throat Hospital, St. Luke's Hospital and the Fifth Avenue Hospital (1926-36) in New York City contain only twenty cases of mediastinitis following rupture of the esophagus which have been recognized and listed as such.

Considering the large suburban area from which these hospitals draw patients and the number attending the broncho-esophagological clinics, one is impressed by the small number of cases.

*Read before the Eastern Section of the American Laryngological, Rhinological and Otolaryngolical Society, Boston, Mass., January 11, 1939.

If this is true in New York it must be doubly true in less populated areas.

There are no records to indicate the number of perforations or ruptures of the esophagus which were not followed by mediastinitis. There must have been some perforations, which were not followed by infection. Also some which made a spontaneous recovery.

CAUSE OF RUPTURE—ESOPHAGEAL RUPTURE IS CAUSED BY:

1. Foreign bodies. Those with ragged edges or sharp points, such as fish bones, chicken bones, dental bridges, pins of various shapes, and thumb tacks, are the most common perforating objects. Perforation from these objects may occur at once by their very nature, may slowly erode through when they are long in situ, or the perforation may occur during their removal. These are perforations which the advance in skill in esophagoscopy cannot alter except in a small percentage of those perforations caused by unskilled removal.

2. The second group of perforations of the esophagus are those caused by instrumentation, first in the hands of the unskilled, by the blind passing of bougies and probangs through unrecognized strictures and over foreign bodies of all types and secondly those caused by instrumentation by the endoscopist himself either in making a false passage along the side of the esophagus or perforation by biopsy.

3. The third group are spontaneous ruptures accompanying malignancy. The esophagus may be perforated in any part of its course. The most frequent site of perforation is at the upper end, at the cricopharyngeus muscle, for it is here that most foreign bodies lodge and it is here that instrumentation meets its first resistance.

The cervical periesophageal abscesses originating here are easily observed and easily approachable surgically. They are at first confined to the cervical region in the area between the esophagus and prevertebral fascia posteriorly or the trachea and esophagus anteriorly. They only later descend into the visceral space. The perforations occurring in the lower parts of the esophagus enter directly into the mediastinum in the region of the large blood vessels and heart. This type causes immediate complications, presents a more difficult surgical approach and are better handled by the thoracic surgeon.

Briefly the diagnosis of a cervical periesophageal infection is made by (1) the suspected or observed perforation of the esophagus by a foreign body, instrument or malignancy; (2) the marked collapse of the patient observed at the time of perforation. This

collapse is much greater following direct perforation by instrumentation or by biopsy than by a gradual erosion of a foreign body; (3) pain, tenderness, and swelling over the area; (4) inability to swallow; (5) absence of dyspnea unless a pneumothorax has occurred; (6) increased leucocyte count of 15,000 to 23,000, usually the higher; (7) sudden rise of temperature though seldom higher than 103 degrees; (8) definite evidence by roentgen examination of a widening of the prevertebral or posttracheal space; (9) emphysema which may be readily ascertained by palpation and observation or may only be recognized by roentgenological examination.

The roentgenogram is the determining factor in diagnosis and aids in differentiating between a simple cellulitis which might recover and abscess formation with a bubble of air. Daily roentgenograms should be made following any known perforation.

Roentgenograms taken with the film at a distance of the shoulder from the veretebral column are not as instructive in diagnosis as the film which is placed close to the neck.

Dr. Fred Law has devised a very simple hanging frame, used with the patient in an upright position. This has a revolving circular plate of wood about one and a half feet in diameter from which about one-third of the circumference has been cut off in a straight line. On this altered revolving circular plack is clamped the x-ray film. In this position the plate comes close to the neck, the flat edge on the shoulder. By rotation of the plack the plate comes into position well down over the lower part of the neck and close to the skin.

The question of how to proceed after the development of a cervical esophageal infection has been considered by many writers. There appear to be three groups. (1) Those who advocate early external surgical drainage in all cases of cervical periesophageal involvement. (2) Those who advise watchful waiting before interference. (3) Those who advocate intraesophageal drainage.

The technique of the anterior approach is practically the original operation of Marschik. An incision is made along the medial border of the sternomastoid muscle. The length and position of the incision is determined by the fact that the esophagus begins at the level of the sixth cervical vertebra. The muscle and the cartoid sheath are identified and together they are retracted to the side. The omohyoid muscle is not severed unless necessary. The lobe of the thyroid is revealed by blunt dissection and is retracted medially. The middle thyroid vein and, if necessary, the inferior thyroid artery are ligated

to give greater deflection of the thyroid gland. This may be avoided if the gland is small. This exposure gives a direct entrance to the infected area. The outline of the larynx, esophagus and prevertebral fascia is readily seen, the vertebral processes can be readily palpitated with the finger. The abscess can be opened easly by blunt dissection or by finger dissection. The esophagus is lifted from the prevertebral fascia and two drainage tubes placed to the infected area. Frequent suction should be applied. The right side is the easier approach because of the position of the dome of the right pleural sac as compared to the left. The esophagus is in contact with the pleura on the right side in the middle section only. On the left side it is in contact with the pleura in the cervical region and also at its lower end just before it enters the stomach. The structures to be avoided are the right recurrent laryngeal nerve and the dome of the pleural sac. If the operation is on the left the thoracic duct must be avoided. Care must be taken to pack off the fascial spaces between the carotid sheath, trachea and esophagus down to the vertebral column, thus blocking as much as possible the entrance of infection into the lower mediastinum.

In those cases in which the infection has passed into the lower mediastinum, a drainage tube may be passed behind the esophagus to this area. The tube must be passed gently and the operator's and interne's finger should not be used too freely in this area or the pleura may be ruptured. After a periesophageal abscess has been located and opened, the less finger manipulation in and around the abscess the better. Nature often does a certain amount of walling off even in an open area as is found here.

In the posterior approach the skin incision is made behind the sternomastoid muscle. In this area practically all the branches of the carotid artery and internal jugular vein are avoided.

The head is turned to the opposite side. The fascia is divided behind the sternomastoid muscle opposite the abscess, and by blunt dissection the abscess is reached by passing through the second layer of fascia behind the carotid sheath directly to the infected area. The only structure to avoid in this approach is the cervical ganglion which is attached to the prevertebral fascia. The omohyoid is not necessarily severed. The brachial plexus may be avoided by keeping above the omohyoid. This approach is quite direct and simple and may deserve more use than has formerly been the case.

Dr. John Lore has found in his anatomical studies of the neck that fluid in the area between the esophagus and trachea have a

Fig. 1.

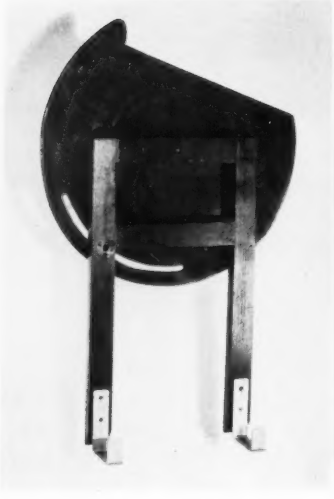

Fig. 2. Front view of Dr. Law's x-ray holder for Roentgenograms of the cervical region.

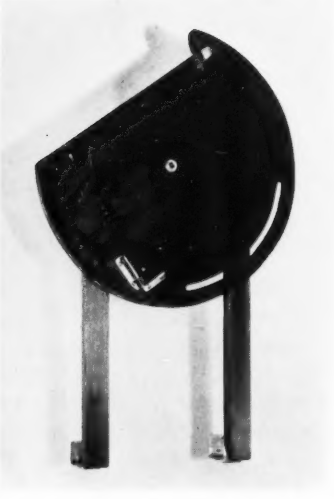

Fig. 3. Back view of Fig. 2.

tendency to point in front of the carotid vessels and sheath anterior to the sternomastoid muscle. Fluid in the region between the esophagus and the vertebra has a tendency to point behind the carotid vessels and sheath posterior to the sternomastoid muscle.

He also made the observation that infection anterior to the esophagus first shows tenderness anterior to the sternomastoid muscle, and a posterior esophageal infection first shows tenderness posterior to the sternomastoid.

These observations in the early diagnoses of periesophageal infection might determine definitely the choice of anterior or posterior surgical approach. It would seem logical to open these abscesses through the place in which they tend to point and through an area which is already infected than through uninfected tissue.

Both of these operations may be done under local anesthesia. Intratracheal anesthesia has been used with good results. A feeding tube should at once be placed through the nose and the esophagus in all cases of suspected rupture of the esophagus. This may in many instances prevent the development of an abscess. After operation the tube should be left in place until healing has been accomplished. The longest time the tube was in place in this series was twenty-six days. There was no trauma to the larynx from the feeding tube in any of these cases. The patient should be put in the Trendelenburg position. Careful after-care is essential. Fifteen of these cases were operated upon by the anterior approach. One was operated upon by the posterior approach.

The following case illustrates the result of early surgical interference.

REPORT OF CASES

CASE 1.—I. G., male, 50 years of age, was admitted to the hospital November 15, 1937, with the history of having swallowed a chicken bone. The foreign body was removed on the day of admission. The following day the patient had a temperature of 102 degrees, pain in the neck and some difficulty in swallowing. The roentgenogram showed some widening of the prevertebral space in the cervical region. The following day emphysema could be palpitated. Temperature 103 degrees. There was more discomfort in swallowing and a second roentgenogram showed an increase in the prevertebral cervical thickening. This area was opened by the anterior approach. Two drainage tubes placed in the wound and a feeding tube placed through the esophagus to the stomach. This patient made an uneventful recovery.

The following case illustrates an attempt at intraesophageal treatment.

CASE 2.—A housewife, 58 years of age, was admitted to the hospital January 20, 1932, complaining of pain in her throat and back, fever, and expectoration of pus. Her present illness dated back to May 27, 1931, when she swallowed a fish bone, which stuck in her throat. She was seen at another hospital, where an x-ray was taken which showed a suspicious shadow behind the esophagus about the level of the arytenoids. The patient did not return to the hospital and was confined to her bed at home with high fever, pain in back and neck, and dysphagia for a period of one month. During the following eight months she had intermittent attacks of pain in her throat, fever, and difficulty in swallowing, which would last for several days, to be followed by a sudden expectoration of one-half to three-fours of a pint of pus, with almost complete relief of symptoms. After two or three weeks of apparently normal health the above sequence of events would be repeated. On admission she was at the hight of the reaction of a very severe attack and had expectorated about one-half pint of pus without the usual relief.

Physical Findings: An acutely ill, feverish, dyspneic, dehydrated middle-aged woman, with a foul breath, complaining of pain in her throat. There was a red, bulging mass in the midline of the posterior laryngopharynx, with pus and mucus obstructing a good view of the larynx. Temperature was 103 degrees and rose to 104.8 degrees.

Laboratory Data: White blood count 17,000, with 80 per cent polymorphonuclear cells, 10 per cent lymphocytes. Sputum was negative for tuberculous bacilli. Roentgenograms of the chest and neck were obtained which showed evidence of an inflammatory process in the upper mediastinum and retroesophageal space.

Course: Shortly after admission the patient expectorated 250 cc. of yellowish pus, which was followed by a fall in the temperature and relief of the symptoms. Repeated esophagoscopies were done and pus drained from a large pocket from the posterior wall of the esophagus just below the cricopharyngeus. It was felt that this improvement would, as in the past, only be temporary and gastrostomy followed by incision and drainage of the upper mediastinum was proposed. Three days after admission a Janeway type gastrostomy was done under local anesthesia and the esophagus was put at rest. Two days later the retroesophageal and superior mediastinal abscess was opened through an incision along the anterior border of the left sternomastoid muscle, retracting the muscle and deep vessels laterally and the trachea and esophagus medially. Cigarette drains were inserted well down into the thorax and the wound packed open with vaseline gauze.

The postoperative course was smooth. Nineteen days after the second operation, lopiodol was injected into the draining neck sinus and a roentgenogram obtained showing the retroesophageal cavity with a communication into the esophagus.

The patient gained ten pounds in weight, and in April she was allowed fluids by mouth and was soon on a full diet. The gastrostoma was allowed to close and, except for a slight sensation of fullness in her throat at the end of a meal, she has been symptom free.

ANALYSIS OF TWENTY CASES

There have been twenty cases of upper mediastinal involvement following perforation of the esophagus observed and treated. The diagnosis of cervical periesophageal abscess was confirmed in all cases

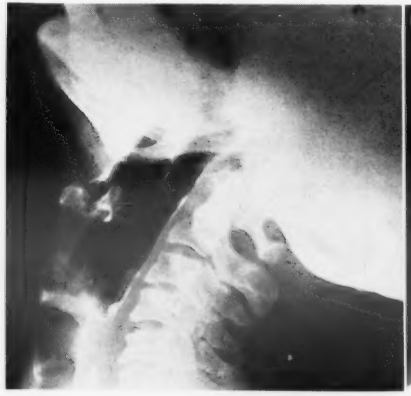

Fig. 4. Case 1. Showing foreign body in esophagus.

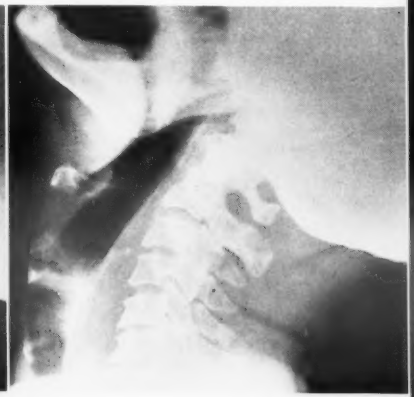

Fig. 5. Second day following removal of foreign body, showing widening of pre-cervical space.

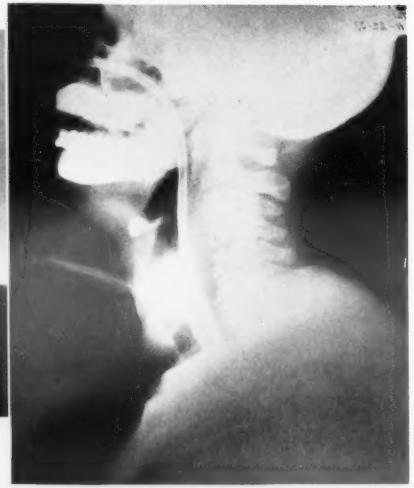

Fig 6. Following external drainage of pre-cervical space, showing feeding tube and two drainage tubes in place.

by roentgenograms. They all had definite widening of the prevertebral space in the cervical region with greater or lesser degree of emphysema.

The perforation of the esophagus was caused by a foreign body in seventeen out of the twenty cases. Of the remaining three cases, one followed instrumentation and two followed a biopsy. The highest temperature in the operated cases was 103 degrees before operation. The white cell count ranged from 15,000 to 23,000. The predominating organism was hemolytic streptococcus, except in one case, which was a Welch bacillus infection.

Sixteen of the twenty cases were operated on and external drainage established, with twelve recoveries and four deaths. Four cases were not operated upon but were treated conservatively or through the esophagus.

Analysis of the four cases not operated upon externally, all of whom died:

Case 3.—E. G., male, 47 years of age. Developed a cervical periesophageal infection following the removal of a foreign body from the esophagus in another institution. This case was treated conservatively with the aid of esophageal aspiration. This patient died.

Case 4.—C. M., male, 70 years of age. Admitted on October 24, with a cervical periesophageal infection which followed a removal of an esophageal foreign body. This case was treated conservatively by esophageal drainage from October 25 to November 29th, when he died.

Case 5.—J. R., male, admitted to the hospital on May 15. He developed a cervical periesophageal infection following the removal of a foreign body. This case was treated conservatively with esophageal aspiration through the esophagoscope until June 5, when he died of a general mediastinitis.

These cases, all of whom died, were treated by private physicians while in the hospital and the records are far from complete. No notes were made as to why an external operation was not done. The treatment they received was evidently the treatment of choice.

Case 6.—A male, eight years of age. Admitted with a foreign body, a dorsal fin bone of a fish, in situ for one week, having caused an extensive mediastinitis. He died before any operation could be done.

It is possible that some of these four cases might have been saved by an external operation. At least under the treatment they received they all died.

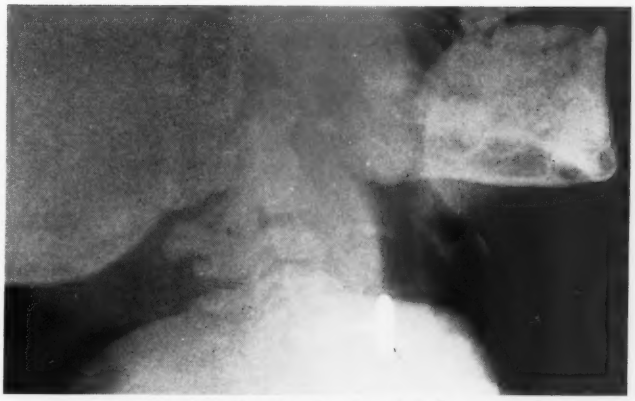

Fig. 7. Case 10. Showing thumb tack before removal.

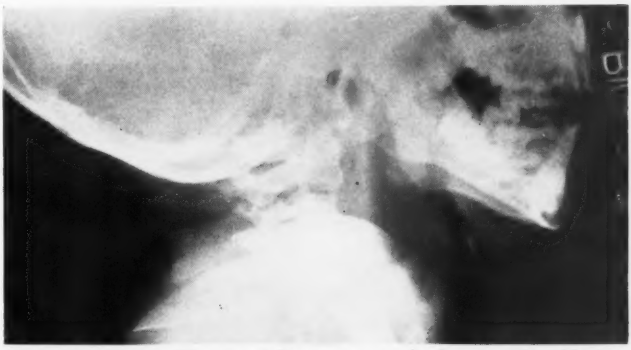

Fig. 8. Case 10. The day following removal of tack. No widening of prevertebral space.

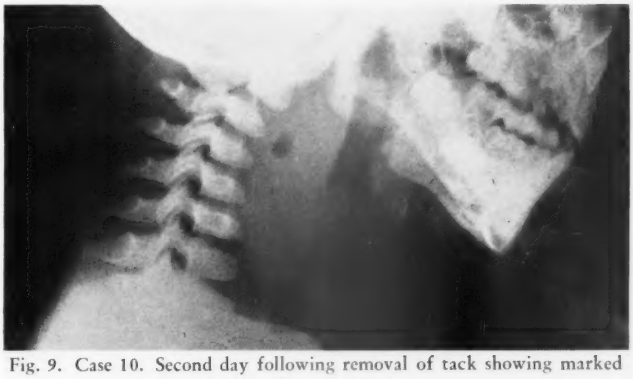

Fig. 9. Case 10. Second day following removal of tack showing marked widening of prevertebral space.

AN ANALYSIS OF THE FOUR DEATHS FOLLOWING OPERATION

CASE 7.—Mediastinitis followed a biopsy on a very extensive malignancy just below the cricopharyngeus in a 65-year-old male.

The posterior mediastinum was promptly opened by the posterior approach and drained. The patient was practically moribund when first admitted due to the malignancy and lack of nourishment. Little else could be expected.

CASE 8.—Female, 45 years of age. A biopsy had been taken just below the cricopharyngeus. A perforation was not suspected and a week's delay occurred before the neck was opened. An extension into the lower mediastinum and pneumothorax promptly occurred. Further surgery was of no avail. If the case had been done earlier perhaps a recovery could have been expected.

CASE 9.—Female, 35 years of age. Admitted to the hospital with the history of having swallowed a chicken bone one week before. She had been under the influence of alcohol for the past ten days prior to admission.

Roentgenograms showed a bone at the cricopharyngeus with a bubble of air attached to its upper part. The prevertebral space was thickened with a bubble of air present; there was some emphysema of the neck. The bone was easily removed. The area around the perforation was opened by the anterior approach and the abscess drained. The culture showed a Welch bacillus infection which spread rapidly in the following two days, resulting in death. This was a case of neglected foreign body with an unusual complication.

CASE 10.—Child, three years old. Admitted to the hospital November 21, 1937, with the history of having swallowed a thumb tack a few hours before admission. The thumb tack on roentgen examination could be seen at the cricopharyngeus, with the point extending posteriorly through the esophagus nearly to the spinus process.

The tack was easily removed by raising the flat part of the tack and grasping the pin part and easing it out of the perforation. A feeding tube was placed in the esophagus at once and replaced twice later. Each time it was not retained even though the child's hands were tied.

His temperature on admission was 98.4 degrees. The following day his temperature was 100.4 degrees, and the roentgenogram showed a periesophageal infection which might subside. The second day the temperature was 102.8 degrees; leucocytes 15,000, and the roentgenograms at this time showed a definite posterior mediastinitus. This was easily opened through the anterior approach. The day following the operation and for four days after removal of the tack the temperature was no higher than 101 degrees. On the fifth day after admission and third day following the opening of the posterior mediastinum the child died of a general mediastinitus and metastatic brain abscess.

Of the four cases out of fifteen who died after operation, in three cases there was either advanced malignancy, an unusual type of infection or a time element which could not be controlled surgically. The fourth case, that of the boy with the thumb tack, makes

one feel that an immediate external prophylactic drainage of the area around any known perforation of the esophagus is good surgery.

It would seem from these cases that early surgical drainage of the infected area is definitely indicated. That the mortality is low if drainage is thoroughly established while the infection is localized in the region of the perforation. That untreated cases are fatal if a real abscess has developed. That external operation and drainage is more thorough and effective than intraesophageal drainage.

One should have the courage to deliberately open the neck on one or both sides and pack off, if not at once in any known rupture of the esophagus, as soon as there is any indication of involvement. Daily roentgenograms should be made, for it is primarily on these that the decision to operate is made. Procrastination means disaster. Boldness means recovery.

CONCLUSION

1. A feeding tube should be placed in the esophagus of any known or suspicioned perforation of the esophagus from whatever cause.

2. Daily roentgenograms should be made.

3. Any rise in temperature, leucocyte count or pain, and particularly x-ray evidence of periesophageal involvement, necessitates an immediate (not the next day) external incision and drainage. The surgical approach is one of choice.

4. Intraesophageal treatment should be used only in the most selected cases.

5. It may be better surgery to deliberately open and pack off any known esophageal rupture before an abscess has developed. If this procedure seems too radical, a very early external operation should be done with the development of any clinical signs of periesophageal abscess.

6. None of these cases had the benefit of sulfanilamide therapy. It is interesting to meditate on its possibilities.

33 EAST 68TH STREET.

XIV

MASTOIDITIS IN CONGENITALLY DEFORMED EARS*

A. T. Wanamaker, M.D.

Seattle

The middle ear and the eustachian tube are developed from the remains of the first brachial cleft, while the pinna and the external meatus are developed from the soft parts overhanging the posterior margin of the same cleft.

"The external ear may be wanting almost entirely, including a complete obliteration of the external canal, or there may be only a slight defect in the pinna, or a membranous obstruction to the canal. All possible degrees and varieties of deformity or deficiency may exist between the above two extremes."[1]

The above is quoted because the two cases presented represent the two extremes.

REPORT OF CASES

Case 1.—Marjorie K., aged 9, complaining of pain back of the left ear, was brought to our office November 29, 1927. Her mother stated that four weeks previously the child developed a sore throat and broke out with a rash that resembled scarlet fever. The rash promptly disappeared. At this time she also had some pain and tenderness in the left mastoid region which lasted two days. However, the temperature continued around 101 to 102 degrees every afternoon and could not be explained by the family doctor, who made a thorough examination, including a fluoroscopy of the chest. He therefore decided, inasmuch as the right tonsil was small but stayed red and there was slight involvement of the cervical glands, to diagnose the condition as glandular fever. When, however, the patient again complained of pain behind the left ear, she was referred to us.

When this child was a small baby, the essayist performed a plastic operation, joining two detached parts to make a better looking ear. When a little over a year of age, Dr. Leede took care of her through an attack of cerebral spinal meningitis in which the meningococcus intracellularis was found.

Examination: Patient rather anemic and underweight. The left ear is rudimentary with complete atresia of the external canal. The mastoid area is red, swollen and tender. Tonsils are small and red. The posterior pharyngeal wall is also red. An audiogram shows normal hearing in the right ear and a loss of 65 per cent in the left.

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*Presented before the Meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Spokane, Wash., January 29, 1939.

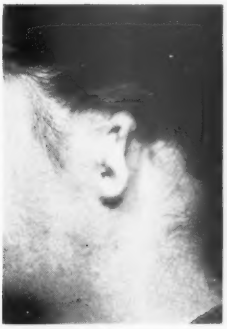

Fig. 1. Case 1.

Laboratory Findings: Hemoglobin, 65 per cent; red blood count, 3,300,000; white blood count, 12,000; polymorphonuclears, 78 per cent; small lymphocytes, 22 per cent; urine, negative.

X-ray shows large, well developed mastoid processes, the left clouded throughout and suspicious of cavity formation.

Operation, November 30, 1927. Subcutaneous tissue thickened and edematous. When an opening in the cortex was made, fully one tablespoonful of thin pus exuded. A large cavity was found involving the tip, and extending to the antrum, the bone was eroded from part of the lateral sinus which was covered with granulations. The zygomatic portion was large and involved, as were also the deep gutter cells. No external auditory canal or drum membrane could be identified. There was a small middle ear that could be probed from the antrum. Culture from the mastoid showed a streptococcus. The recovery was uneventful. An audiogram taken November 29, 1927, showed a loss, by air conduction, of 50 per cent in the left ear. One taken January 7, 1939, showed a loss of 38 per cent by air conduction, and only 5 per cent by bone conduction. She hears conversational voice with a noise apparatus in the right ear at three feet; without the noise apparatus, she hears conversational voice at twenty to twenty-five feet, thus showing considerable improvement since her operation.

Case 2.—Phyllis P., age 15, was referred by Dr. Schutt of Bremerton, March 19, 1938. Mother states that her daughter was born with a deformed left ear, and that six years ago, when she had a cold, this ear began to discharge, and ran for two or three months. There was no further trouble until six weeks ago, when the same ear began to run again. For the past two nights she has had considerable pain.

Examination: The right ear and drum are normal; the left ear is small, just a little more than half the size of the right, and is somewhat deformed. The canal is much narrowed and just anterior to the tympanic ring is a membranous obstruc-

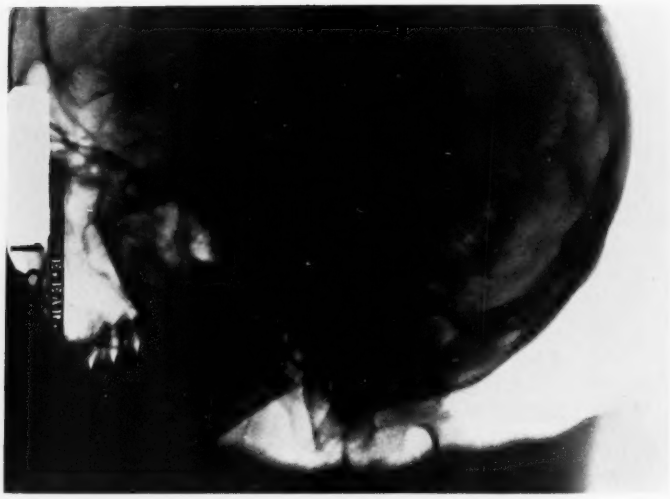

Fig. 2. Case 1. Right ear.

tion with only a pin head opening. A small amount of discharge is coming from the middle ear. The skin behind this ear is red, tender and swollen, so that the ear stands out from the head. All the sinuses transilluminate clearly. She is able to hear whispered voice in the right ear at forty feet, in the left ear at thirty feet. The Weber is referred to the left side.

Laboratory Findings: Hemoglobin, 89 per cent; white blood count, 12,400; polymorphonuclears, 80 per cent; small lymphocytes, 20 per cent; urine, negative; Wassermann, negative.

X-ray shows a large process with well defined cells in the right; on the left side, no cellular structure can be made out; looks like a large cavity.

Operation: A left radical mastoidectomy was performed March 21, 1938. A fistulous opening in the cortex was noted through which came creamy pus. Under the cortex was a large cavity filled with a marble-sized, foul smelling cholesteatoma extending into the antrum. The sigmoid sinus was covered with granulations, the hard palate over it having eroded away. The mastoid antrum was high, small, and irregular in shape. The annulus tympanicus was prominent and overhung somewhat the middle ear. No drum or ossicles could be found. Culture taken at the time of operation showed no growth.

Owing to the small, narrowed canal and small ear, a smaller flap had to be made, but due to the large exposure of the sinus wall, and the presence of the cholesteatoma, an opening was left in the upper part of the wound so that the cavity could be dressed both through this opening and through the canal. The

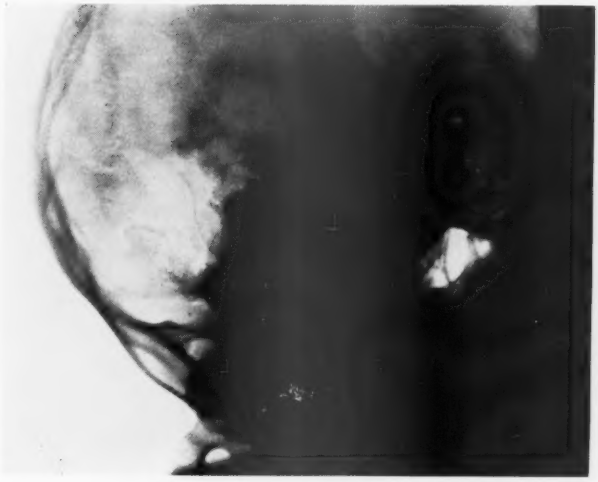

Fig. 3. Case 1. Left ear.

wound healed nicely but there is still the smooth opening back of the ear. This could be closed any time but the hair hides it nicely and it makes it much easier to keep clean. Parents of each child are normal, healthy people who have other normal, healthy children.

DISCUSSION

Beck[1] has found that these patients with a fair degree of hearing will have large mastoid processes, well pneumatized, and well defined internal auditory canals. He cites most interesting autopsy findings on a baby with one rudimentary ear, in which no middle ear was found, the internal ear defective, narrow internal auditory meatus, and no mastoid cell development whatever. In one case he was able to show an intact eustachian tube, but did not attempt to show it in the other cases he reported. My first patient had a large mastoid process with clouded mastoid cells and a fair degree of hearing. In my opinion, the diagnosis was acute suppurative mastoiditis, in which the infection, a streptococcus, traveled from an infected throat by way of the eustachian tube. For my second patient, the diagnosis is not so clear. Her x-ray showed a cavity formation and poorly defined cellular structure, yet her hearing was quite good. The question

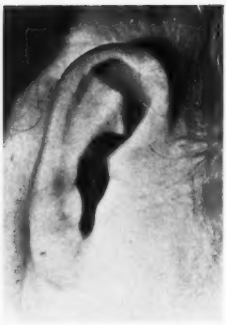

Fig. 4. Case 2. Good ear.

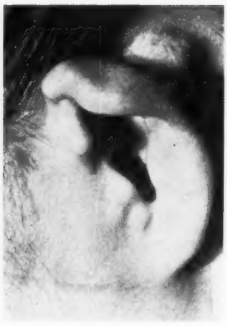

Fig. 5. Case 2. Defective ear.

is, are we dealing with a primary or secondary cholesteatoma. After a review of the literature, one feels that we must be dealing with a primary cholesteatoma.

Goufas,[2] in his paper entitled "Probable Congenital Malformation of Left External and Middle Ear with Chronic Relapsing and Exteriorized Osteitis of Tympanum and Primary Encysted Cholesteatoma of Tympanic Cavity," followed a case where operation was refused for some time, in which there was a history of an external phlegmon and later a recurrence of the same with fistulous formation. There was scar tissue in the external canal forming a partial atresia. At operation there was found to be congenital defect of the middle ear and absence of tympanic antrum and no pneumatization of the mastoid process. A primary cholesteatoma was found. He concludes that the primary cholesteatoma can develop under the influence of an irritation, usually inflammatory, from the included embryological debris, lying in a latent lethargic state, in the depth of the mucus.

Sendulski[3] also had a case of a child with intermittent attacks of pain in her ear with fever, beginning when she was about one year of age, which were relieved by hot packs. At times she had peculiar crackling sounds in the ear. For quite a period she had daily attacks of vertigo. Her parents were nervous and neurotic. One relative on the mother's side had atresia of the ear canal. The Weber localized in the affected, the right, ear. She was only able to hear a loud

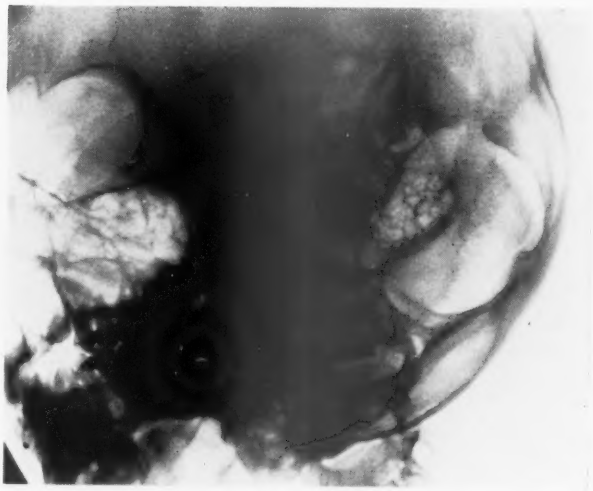

Fig. 6. Case 2. Right ear.

voice spoken in the ear. She was operated at the age of eight, when there was found quite a wide tympanic cavity with some free whitish-gray masses in it. She had a small bony canal. Several peculiar fatty crumbs were found in the mastoid cavity. The mastoid bone was of the diploic type. The fatty crumbs and bone chips were examined microscopically and found to show lymphoid and connective tissue infiltration. The bony canal was kept open by rubber dam until healing took place. A year after operation, the hearing had improved markedly. Micrococcus catarrhalis and staphylococcus aureus were found in the mastoid culture.

The author sums up his case by stating that there was arrested pneumatization, a small antrum with no ossicles and undeveloped middle ear.

Wittmaack[4] believes that the ossicles develop about the fifth month of intra-uterine life, and, therefore, the things found in these undeveloped ears depend upon which month of intra-uterine life arrested development starts. He also contends that in order for normal pneumatization to take place, normal epithelium must be present.

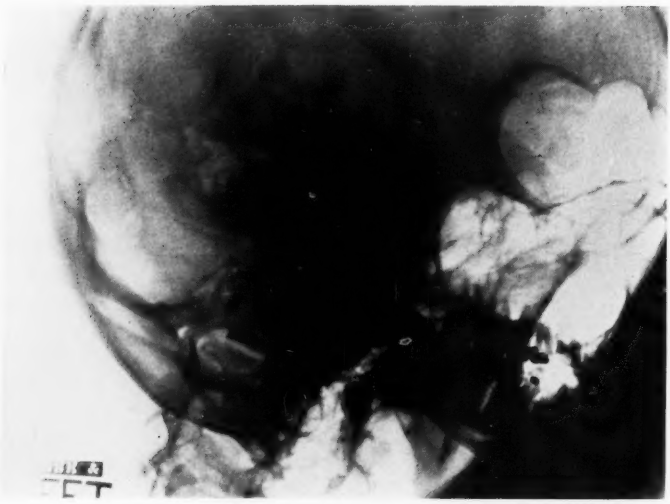

Fig. 7. Case 2. Left ear.

Antonin Precechtel's[5] article is convincing. Several sections of a defective ear, in a new-born child, showed changes in the internal and middle ear. He found epidermal splinters in several places. These splinters have stratified squamous epithelium, on the surface of which the cells are keratinised and desquamated in lamellae and stain deeply. From these findings and the work of Alexander and Benesi he concludes that a primary cholesteatoma would eventually develop.

David-Galatz's[5] article deals with a case of a man 29 years old, upon whose left ear he operated and whose wife later bore a child with a small congenitally deformed right ear. He divides cholesteatomas of the ear into two classes: (a) cholesteatomas without suppuration of the cavity, and (b) those which are accompanied with suppuration of the cavity.

In the absence of a congenital defect in an ear, it is very hard to believe in a primary cholesteatoma. However, Edgar Holmes[7] cites one case in which a man apparently had a primary cholesteatoma on each side. One ear was operated on, the other had not been operated at the time the article was written.

An interesting incident occurred at the American College of Surgeons Clinics of 1927, when Furstenberg presented a child with congenital atresia of the canal and defective external ear, whose hearing he hoped to improve by operation. However, when he reached the antrum he could not identify the parts. Alexander, of Vienna, unexpectedly came into the amphitheater and was immediately asked to give his experience in such cases. He stated that he, likewise, had been unable to do anything in an operative way to improve the hearing, as all these ears showed middle as well as external ear defects.

For an intensive study of cholesteatoma, read R. Wallace Teed's[8] article "Cholesteatoma Verum Tympani: Its Relationship to the First Epibranchial Placode."

CONCLUSIONS

1. Pain in the mastoid region of a congenitally defective ear should call for immediate, thorough examination and an x-ray picture.

2. Discharge from such an ear likewise is a suspicion of trouble in the bone and a probable primary cholesteatoma.

3. Operations only to improve the hearing are useless.

1317 MARION STREET.

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XV

RESTORING FORCES IN THE MECHANICS OF AUDITION*

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Los Angeles

Audition is the result of a reactance to sound and may be separated into a physical and a physiological component. The physical component may be said to include all displacements from the sound source to the activation of the auditory cells where the physiological component begins to terminate in the central nervous system. The accepted interpretation of the physical component assumes that the activation of the auditory cells is an indirect one, and that these cells are nonspecific in their response. This assumption is based on the anatomical fact that the auditory cells show no evidences of a structural difference which might be correlated with the function of a descrete frequency analysis. Accordingly practically all theories believe that the internal ear may be regarded as nothing more than a mechanical harmonic analyzer. The specificity in the response is explained by the location of the area affected in relation to the length of the end organ (frequency analysis); while the intensity in the response is dependent on the amplitude of the reaction in this localized area (loudness). This accepted interpretation is objectionable from a number of points of view, and not the least among them, is the effect which this theory has had on the problem of the deafened individual.

An excellent differential diagnosis between a conduction and a perception deafness was established by Capivaccio before the close of the Sixteenth Centry. A French layman, Guyot, discovered the value of the inflation of the middle ear in 1724; while Clelland, in 1741, employed a tubal catheter which might be mistaken for part of the equipment of a modern otologist. Astley Cooper in 1802 described the incision of the drum membrane with an increased acuity in certain deafened cases. All of these findings were established long before the discovery of the end organ by Corti. Probably the most outstanding fact regarding the auditory apparatus was the establishment of the labyrinth liquid by Cotugna in 1774. This

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finding forever put to rest the old arguments on the Aer implantus, and at the same time threw a monkey wrench into the mechanics of audition from which it has never completely recovered.

Two master-biophysicists, E. H. Weber and Hermann von Helmholtz, are responsible for the more or less generally accepted interpretation of today. One might say that Weber wrote the words which von Helmholtz set to music. The reputation of these two mighty men of science is so great that anyone challenging the validity of their obvious explanation closely approaches a profaning of the family gods. However, it must be remembered that all explanations may be regarded as purely tentative until all related facts have been established. Perhaps the entire problem of hearing has suffered through over-visualization when the relations might have been more properly auditized. It may be well to listen to the reactions in the living ear and use such quantitative measurements as a basis for a proper interpretation of the structural responses.

The title indicates we are considering the restoring forces in the auditory apparatus. This quantity has been treated in a very stepmotherly manner by practically all investigators, and seemingly is almost an unknown quantity among the otologists. My apologies in advance if the treatment is a very elementary one.

When a given medium or object is deformed or is displaced from a position of rest and tends to return to its original form or position, the forces which bring this about are spoken of as restoring forces. For example, a properly hung door has no restoring forces and therefore tends to remain in the position to which it is displaced. A swinging door, however, has restoring forces which tend to shut it once it has been opened. These forces may be located within the medium or object (intrinsic) or they may be situated outside (extrinsic). They may be related to the mass moved as a mass restoration, or they may be molecular in character as illustrated in sound displacements. The difference between a mass and a molecular reaction may be shown in the behavior of two familiar objects. If one pushes one's finger into a mass of modeling clay, the clay remains displaced, but if one does the same thing with a piece of sponge rubber, the rubber returns to its original form. This is a mass reaction and indicates that the rubber is more elastic than the clay. However, if these two objects are placed upon a resonator box and the stem of a vibrating fork is applied to each, we then hear that the sound displacements pass more effectively through the clay than

through the rubber. This means that so far as the molecular reactions are concerned the clay is more elastic than the rubber.

If one takes a glass graduate of some 50 cms. length and sounds a 256 fork over it, the resonant response will not be critical until sufficient water is added to make the column of air some 32 cms. long. This is true because the phenomenon is dependent on a reflection back of the sound waves and the creation of a standing wave with a node at the water surface and a loop at the open end of the tube. Accordingly, a closed-end resonator speaks to one-fourth of the wave length and this is dependent on the speed of the sound in air. Now if a few drops of carbon tetrachloride be added, the evaporation of this liquid contributes to the weight of the gas but does not affect its elasticity. Accordingly, the speed of the sound will be slowed down and more water must be added to arrive at the critical one-fourth wave length. This simple experiment demonstrates the standing wave, and at the same time indicates that air vibrations do not effectively pass into water. This fact was known in the middle ages and was responsible for the assumption of an Aer implantus, because it was argued if the internal ear had been filled with the liquid, described from time to time, it would have been relatively deaf to air sounds. The logic is quite correct, and the internal ear as an immersion receptor is relatively deaf to air sounds. I chose to call this an acoustic insulation of the labyrinth.

The answer to the physical riddle of Cotunga must therefore be found in the addition of some agency which will drive the air vibrations into the labyrinth and give this relatively deaf internal ear something to listen to. This was what Weber attempted to do in his interpretation of the middle ear apparatus as, what might be termed, a resistance matching transformer. Suppose one places a glass of water on the resonator box and then immerses the stem of a vibrating fork, little effect is noted. However, if one widens the fork stem to match the difference between the resistance of the water and the stem, then the resonator speaks quite loudly. It is well to bear in mind that the columellar and the stapes footplate is many times the area of the narrowest part of the sound conduction apparatus. But when Weber applied his transformer idea over to the labyrinth, he abandoned the molecular quantity with which he had started out and conceived the present accepted idea of a mass movement in the labyrinth liquid.

We are now prepared to consider the only theory of the function of the cochlea as a mechanical harmonic analyzer which might explain an extrinsic activation of the nonspecific auditory cells.

The theory is that of Weber-von Helmholtz brought up to date through the addition of the selective weighting in the liquid of the scalar columns. The sound pulse entering the external auditory canal displaces the drum membrane inward, carrying with it the attached ossicular chain. The handle of the malleus is somewhat longer than the long process of the incus and this results in the familiar bent-lever theory of sound conduction where the motion is decreased and the power correspondingly increased. The stapes footplate is applied to the labyrinth liquid, which is regarded as incompressible contained within the rigid otic capsule. Under these postulated conditions the labyrinth liquid could not accept the vibratory thrusts of the stapes unless some compensation area was available which would permit the shifting of the labyrinth liquid as a whole. This compensation area is found in the round window membrane. Accordingly, as the stapes footplate is displaced inward, the liquid of the scala vestibuli moves toward the helicotrema, while that of the scala tympani passes toward the round window which moves outward. The differential pressure created displaces the interposed membranous cochlear duct downward. The entire system from drum membrane to round window swings as one unit to the pressure of the applied sound pulse, and now the restoring forces in the system must go into operation to swing this entire unit back again so that it may faithfully follow the varying displacements in the sound vibrations heard. This is the explanation offered by Weber to produce the transverse vibrations in the basilar membrane, but which, as von Helmholtz correctly indicated, did not solve the problem of the frequency analysis.

Von Helmholtz added to the Weber interpretation in his familiar pianoforte or harp theory. He conceived the basilar membrane as composed of a series of transverse fibers which supported the organ of Corti. He believed that these individual fibres reacted to sound by reason of resonance or sympathetic vibration. Accordingly, the shorter fibres located near the two windows would react to high frequencies, while the longer fibres near the helicotrema would respond to low frequencies. Each fibre would therefore stimulate the applied auditory cells and this would be translated by the brain into terms of pitch.

There are, however, objections to this interpretation. The resonant response in a string is dependent on the length, the tension, and the weight. The alleged strings composing the basilar membrane did not show sufficient differences in the length, tension and weight to produce out of them an eleven octave harp. Clearly then the

basilar membrane did not show structural differences to account for the range of specific frequency responses any more than did the auditory cells. However, an attempt was made to compensate for the anatomical deficiences in the explanation by adding a variable factor which might tune the strings. This variable factor was located in the scalar liquid because, on account of the interaction between the two windows, there would be less weight to be moved when the short strings were vibrated and more weight involved when the longer strings near the helicotrema were activated. It is obvious from this brief description that not only does the auditory apparatus move as one unit from the drum membrane to the round window but that the selective weighting in this conduction system is responsible for the critical tuning of the extrinsic activation by reason of the transverse vibrations in the basilar membrane. Curiously enough, the very system of mechanics employed by the Bonnier-ter Kuile-Max Meyer theory to account for a selective intensity reaction, is also being employed to explain the specific frequency analysis. Further, neither the theory of the length of the end-organ as a function of the intensity nor the length of the end-organ as a function of the frequency, note the importance of the restoring forces essential to the successful operation of either explanation.

Many mistakes have been made in the applied mechanics of audition. When I pointed out in Copenhagen that the evidence indicated the drum membrane and ossicular chain was the only efficient route in the conduction of air vibrations to the cochlea, there was a storm of protest. Alexander, in a summary which appeared some years later, did not even consider the objections of sufficient importance to mention. The audiometers at this time showed the typical conduction curve as a low frequency deafness. Later I pointed out that conduction deafness merely brought to light the normal sensitivity of the internal ear as a function of the frequency and that this was being mistaken for frequency limitations in the conduction apparatus. Now I note that all audiometers give the typical flat curve for conduction deafness, and that cases with appreciably greater losses in sensitivity for high frequencies are definitely related to middle ear disability.

I stated years ago at the Montreal meeting that we do not require animal experimentation to refute the theory of a selective weighting. This is true because cases with liquid in the intact middle ear are extremely common. Under these conditions the round window membrane is loaded and the selective weighting relations in the cochlea are very seriously disturbed. Such cases should, therefore,

have a false hearing throughout the entire frequency range, but I have seen no such cases, and Politzer says that false hearing does not occur under these conditions. Some recent cases reported by Crowe and Polvogt are interesting in this connection. "There was a dehiscence in the bony septum between the middle and apical turns in six of the seventeen cochleæ. This anomaly was described by Alexander, who called it scala communis cochleæ. According to some theories of hearing, a scala communis which establishes a free communication between the scala vestibuli and the scala tympani, should impair the hearing. The location of the defect in our cases should theoretically impair the hearing for low tones, but the audiometer test showed normal threshold acuity for all tones in every patient."

We may, therefore, dismiss the selective weighting through the applied scalæ and delete from the picture the only factor which might make the von Helmholtz theory of resonant analysis in the strings of the basilar membrane act as a mechanical harmonic analyzer. This would be true even if we disregarded what we saw, and believed that there really were such strings. This, then, leads us back to the source whence all these blessings flow—the Weber interpretation, and in particular to the restoring forces in this system—a factor which is not even considered in the recent comprehensive book on hearing by Stevens and Davis.

In general terms, the restoring forces in the auditory system may be located in several places; the drum membrane, the trapped air of the middle ear, the intrinsic muscles, the ligamentous attachment of the ossicles, and in particular the elastic annular ligament of the stapes and the elastic tissue of the round window membrane. This is true, because the restoring forces are definitely related to the elasticity and the weight of the system which is responding.

For example, the removal of the drum membrane might operate in two ways: first, by reason of the loss in the weight and the elasticity of the membrane itself; and second, because the push-back of the air trapped in the middle ear is eliminated. Yet the only effect noted is a loss in sensitivity due to the loss of the drum membrane surface, and no false frequency discrimination is found. The tensor tympani muscle seemingly does not react to sound in the human being, but the evidence furnished by Luescher indicates that the stapedius does respond even to sounds of relatively low intensity. Contraction of the stapedius should affect both the weight and the tension in the apparatus and therefore have a decided effect both on the frequency analysis and on the intensity. Accordingly, if an individual is tested in a dead room under conditions where one ear

is masked against no masking, the minimum acuity curve should give us definite information on the quantitative effect of the increase in tension through the contracted stapedius muscle. We find it has no effect. This leaves us only the ligamentous attachments of the outer ossicles and we compare the curve of acuity of a case in which the drum membrane and outer ossicles have been removed, with a normal curve and find the only change is in the intensity and that the frequency discrimination is normal. This leaves only the elastic ligamentum annulare of the stapes and the elastic membrane of the round window to be considered. How elastic is this tissue and how well is it adapted to act as a restoring force in the displacements brought about by sound vibrations? If one places a piece of the ligamentum nuchæ of the ox on a resonantor and applies the stem of a vibrating fork in period with this resonator, one finds that the tissue behaves much like a piece of sponge rubber. Accordingly, while it is poorly adapted to act as a restoring force, it is a most excellent insulation of the stapes footplate against the discharge of the vibratory displacements into the otic capsule. The responses in the elastic tissue can therefore be only to relatively slow excursions.

Suppose we contrast the acuity of hearing in a case where the outer ossicles are wanting but the stapes area is moveable with one in a similar condition where the stapes area is fixed. In the first case all mass movements in the cochlea are suppressed by reason of cancellation, and in the second case, the fixation of the stapes suppresses all possibility of the to-and-fro displacements in the scalæ. Both types of cases should be quite deaf to air and bone conducted sounds, but the bone acuity tells us that in neither case have the conditions been violated under which the cochlea analyzes the sound vibrations.

The physicist von Békésy has interested himself in the cochlear mechanics under conditions where the outer ossicles have been lost or removed. His Fig. 1 indicates the accepted mechanics as I have stated them, and his Fig. 2 shows the reactions where the drum membrane and outer ossicles are wanting. In Fig. 2 it will be noted that the sound pressures are applied only to the round window and that this is regarded as the portal of entry for the air vibrations. Under these conditions some additional vents become operative. These vents are the blood vessels, the ductus endolymphaticus and the ductus perilymphaticus. He holds that the upper system of vents is more effective in permitting the displacement of the liquid than the lower vent. As a result, the basilar membrane now responds in opposite phase, as indicated by the arrows, and the patient reports this change in phase by reason of a change in direction of the sound heard.

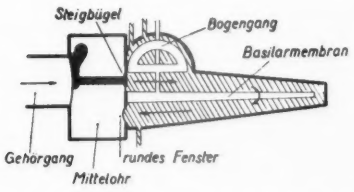

Fig. 1. Schematic representation of the organ of hearing. (After von Békésy.)

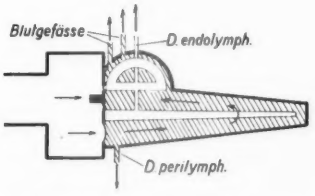

Fig. 2. Direction of flow of the Cochlear fluid in the absence of the membrana tympani. (After von Békésy.)

Placing a cotton pellet against the stapes area brings about a re-reversal in the responses in the basilar membrane and is again indicated by the subjective response.

The explanation leaves much to be desired. It seems curious that the additional vents should remain quite inoperative until the drum membrane and outer ossicles have been removed. It also remains to be proved that the stapes area does not vibrate to the air sounds applied to it in the same phase as that at the round window. We do not need to speculate about this problem, because the evidence is already at hand. Every case of progressive conduction deafness will arrive at a time when, be these venting factors what and where they may be, the amount of vibrational thrust through the stapes will exactly balance those through the round window. Then this case will go completely deaf some time before the stapes is absolutely fixed. So far as I am aware, no such case has ever been reported, although it should be the common experience of all otosclerotics. Again may we be permitted to ask, if the sound pressure pushes the labyrinth liquid up into these vents, where are the restoring forces which push it back again?

It appears that the Weber conception of internal ear mechanics is based upon premises which are individually and collectively untenable. Perhaps this may account for the fact that no theory of hearing based upon the Weber conception fits the experimental evidence

During the past few years there seems to have been a revival of learning among the otologists and they are beginning to invade a territory which properly belongs to them. There have been a number of ventilation operations performed on the labyrinth to improve the hearing, and these operations have seemingly done in part the purpose for which they were intended. But had these otologists clearly understood the physics of the accepted interpretation of auditory mechanics, they would never have attempted the operation. This is true because according to the accepted interpretation all these cases should hear much worse. Can it be possible that we must go back to the constructive suggestions of Astley Cooper in 1802 and to Toynbee in 1860 for the only advances in the direct treatment of deafness?

We have found that the only restoring forces in the auditory apparatus are those slowly reacting ones which cannot respond to sound vibrations. We may therefore associate them with the shock-absorber function of the middle ear apparatus which has been mistaken for the mechanism of sound conduction and sound analysis. Herein we may find an explanation why the sound conduction system is so markedly aperiodic in its responses because the only restoring forces involved are those which are intrinsic to the various media of conduction. We do not know how we hear, but neither do we know how we detect the odor of garlic from that of roses. What we hear is probably nothing more than the sound pulses in the labyrinth liquid and may assume a specific reaction in the auditory cells to these sound pulses which perhaps pass through them. This statement implies that the physical component in audition ends with the development of the sound pulses in the labyrinth liquid where the physiological component begins with the specificity of the auditory cells themselves.

May I, in closing, attempt to visualize the factor with which we are dealing because it was the amplitude and the power value in sounds which started me on this problem some twenty-five years ago. The displacements in the air of the external canal at minimum audition in the frequency range of greatest sensitivity is $10^{-9\text{th.}}$ cms. and the power value at 0 db. is $10^{-16\text{th.}}$ watts. This means when

the swing in the air of the external auditory canal has attained the dimensions of a white blood cell, the intensity of the sound heard is 120 db. which is about the limit which can be tolerated. The $10^{-16th.}$ watts means that if the auditory system swings as a whole and weighs one gram, then the energy consumed in a 100-watt lamp will vibrate a fleet of 30,000,000 thirty-five thousand-ton battleships. In spite of all of this we are still talking about the mechanical advantages in the umbo; the leverage action in the ossicles; the tilting action in the stapes; the displacement of the liquid of the scalæ in opposite phase; the transverse reaction in the cochlear duct; and the round window membrane as a compensation opening essential to audition. At the same time, the conduction deafened individual is only deafened because his immersion receptor is relatively insensitive to air vibrations, and although his internal ear be quite normal, all we appear able to do is to supply him with a hearing aid and encourage the study of lip-reading. If these are the only constructive suggestions which have developed through acceptance of a theory of cochlear mechanics, the time is ripe for the consideration of other possibilities.

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XVI

PERTINENT FACTS RELATING TO CHRONIC PANSINUSITIS*

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When I decided to discuss the subject of chronic pansinusitis I did so, realizing that I had nothing original to offer, and that I could, therefore, only give the results of my personal experience, and perhaps, to some extent, stress what seemed to be the fundamental problems which confront us in our daily examination and treatment of these cases. That chronic pansinusitis is of frequent occurrence will not be denied, and since the paranasal sinuses are linked together in a physiological, etiological, pathological and bacteriological manner, it follows that disease limited to one cavity is a rarity.[1] This is true of acute as well as chronic inflammation of the nasal sinuses.

There has always been a doubt in my mind whether the mucous membrane of the ethmoid sinuses, for instance, ever returns to a normal state following a severe nasal sinus infection associated with such diseases as scarlet fever, measles, influenza and the like. There are several good reasons why resolution following an infection in this region is difficult. In the first place, a laterally displaced edematous middle turbinate covering the ethmoid floor, in the shape of a lid, will prevent ventilation and drainage. The ostia of the ethmoid sinuses become swollen, secretions are retained, and there is a great tendency for the development of a latent disease. When latency occurs, recurrent infections soon follow with the possibility of involvement of other sinuses. This is one way in which a chronic pansinusitis may develop. Following an acute infection, a latency may also be established in the maxillary sinuses except, in this instance the discharge, when it does occur, is usually more profuse and there is a tendency for the latent antrum disease to give rise to a systemic derangement of some kind.

When there is present a chronic disease of various cavities it does not necessarily imply that the same type of inflammation has

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affected all of the sinuses. We have observed on numerous occasions that a suppurative disease of the antrum, for example, was associated with a serous inflammation in the ethmoid, and that suppuration within the ethmoid sinus secondarily involved the frontal sinus with a serous inflammation. It is equally important to remember that a posterior ethmoid infection may cause sphenoid involvement, and that in many instances the pathological changes are not present within the sphenoid cavity, but instead there has occurred hypertrophy and edema of the mucous membrane of the anterior wall of the sphenoid with closure of the ostium. It is furthermore worthy of note that the antrum cavity often acts as a reservoir for the pus from above, and that the removal of the infection higher up may satisfactorily care for the disease in the antrum. Onodi was the first to show that there may be recesses in the sphenoid sinus called by him "recessus maxillaris," and also recesses in the antrum termed "recessus sphenoidalis."[2] The presence of such recesses brings these two cavities in close proximity, so that infection of one easily causes involvement in the other. As a matter of fact, these recesses may be so well developed that the posterior choanæ become narrowed.

I propose to touch briefly on a few of the symptoms pertaining to chronic pansinusitis. The most constant symptoms common to almost every case are headache, nasal discharge, lack of concentration, and a feeling of pressure in the head. The patient states that he feels "as if a tight band is stretched over the forehead." Headache is not always caused by the sinus infection per se, but is often the result of pressure of the middle turbinate against the lateral wall of the nose. Relief of this pressure by infracting the middle turbinate and cauterizing the lateral wall of the nose opposite the middle turbinate with a concentrated solution of trichloracetic acid often relieves the headache.

In some instances the cause of the headache is hard to determine because of lack of clinical findings. An anatomical malformation within the nose or sinuses is often responsible for the same. A high deviation of the septum, closure of the nasofrontal duct from whatever cause, tortuosity of the duct as the result of a displaced infundibular cell, a large bulla and uncinate cell in close apposition, aberrant inter-frontal cells, laterally displaced orbital cells, closure of the ostium of the frontal sinus by a thickened mucosa on the floor, marked deviation of the inter-frontal septum causing blocking of the ostium, and a well-developed processus frontalis are some of the anatomical reasons for headache.[3] It may happen after doing a radical antrum and intranasal ethmoid operation that the headache,

nasal discharge and mental hebetude continue to exist. This cannot be blamed alone on the affected frontal sinus, for this cavity may be but mildly involved. An anatomical variation is more apt to be the cause.

During an acute exacerbation of the chronic sinus disease, the headache and feeling of pressure are aggravated. When discharge of mucus or pus is free there seems to be less toxic absorption, and the patient, as far as the general health is concerned, feels better. When the disease once more assumes the latent stage, systemic invasion more readily occurs.[4] The improvement in health with gain in weight and return of appetite following a complete operation for chronic pansinusitis impresses one with the fact that the systemic derangement caused by the sinus disease was responsible for many of the symptoms.

The diagnosis of chronic pansinusitis is apt to be difficult in some cases. It is easy to arrive at a decision when the nose is filled with polypi and the skiagram shows an opacity of all sinuses, or if pus is found coming from the region of the nasofrontal duct and olfactory fissure, and antrum lavage with lipiodol instillation definitely shows an involvement of this cavity. It is, at times, impossible to say whether the pus one sees in the region of the nasofrontal duct is coming from the frontal sinus or from an upward or laterally displaced ethmoid cell. Mild cloudiness of the frontal sinus, as seen in the skiagram, is not conclusive, inasmuch as the opacity may be the result of closure of the nasofrontal duct with absence of air in the frontal sinus which may be the cause of the hazy skiagram.

It will be remembered, I stated that a suppuration in one cavity may be associated with a serous inflammation in an adjacent sinus. With this thought in mind, it behooves us to examine more carefully the ethmoid region before completing a radical antrum operation. Polypoid changes in the region of the uncinate process, olfactory fissure, anterior end of the middle turbinate and the floor of the ethmoid are characteristic of ethmoid sinus disease, but it does not follow that absence of such changes means there is present no ethmoid involvement. Mild pathological changes of the ethmoid may be present and remain unrecognized unless the floor of the ethmoid is examined. The floor may be viewed easily with median rhinoscopy, by using a long-bladed Killian speculum and infracting the middle turbinate toward the septum. This method of examination should, in my opinion, always be used in the routine examination of the sinuses. If this is done, mild polypoid changes will often be

seen, and occasionally droplets of pus will be noticed exuding from the ethmoid ostia. In like manner, the sphenoid area may be more thoroughly examined by infracting the middle turbinate toward the lateral wall of the nose. It is always easy to decide that the frontal sinus is involved when there is a marked involvement shown per skiagram, or when there is external evidence of disease within this cavity. It is impossible to say, however, whether this sinus is affected when the skiagram is mildly opaque or entirely negative in appearance. We have seen frontal sinuses in which a negative interpretation of the skiagram was made, and where, at the time of the external ethmoid operation, a serous inflammation with a fair amount of edematous tissue was found.

It is important to keep in mind that many patients with chronic nasal sinus disease of the serous type are allergic individuals. Continuous allergy prepares a fertile soil for bacterial invasion, and as a result of many insults the mucous membrane of the sinus becomes thickened and polypoid in character. In these cases, desensitization alone will many times not suffice to bring about relief. Surgery is often the ultimate solution to the problem. Surgery, however, should not be attempted until an allergic diagnosis and treatment has been provided. Repeated microscopic examination of the nasal discharge in these cases is very enlightening. It is interesting to note the diminution in the number of neutrophiles and the increase of eosinophiles as the acute manifestations subside and the mucous membrane assumes once more its chronic allergic state. Hansel has rightly laid great stress on this point.[5] It is self-evident that a long standing allergic sinusitis predisposes the patient to many acute nasal sinus infections.

There seems to be a sharp diversity of opinion among rhinologists regarding the proper surgical approach to a case of chronic pansinusitis. The ultra-conservatives content themselves by doing a submucous resection of the septum, making a window under the inferior turbinate and attacking the ethmoid sinuses intranasally. The personal view of the operator will, as a general rule, determine the extent of operative interference. There are some who are convinced that the proper surgical procedure is to remove all diseased tissue, in a single period, from every involved sinus, and many times they are right. If this method of approach is the one universally adopted, it will be found at the time of the operation that occasionally the pathological changes are mild in character, and that there is an absence of anatomical variations. If such a coincidence arises, we will have to admit that in all probability a more conservative oper-

ation may have sufficed. There are other rhinologists who are content to do a radical antrum and intranasal ethmofrontal operation, wait a reasonable time if no emergency arises, and if a good result is not obtained, to proceed by way of the external route. We have learned from experience that it is safer to proceed with the external ethmofrontal operation, if while operating intranasally, pus is seen flowing from the region of the nasofrontal duct, and if, in sounding the frontal sinus, pus exudes from the duct each time the sound is removed. If we proceed with the external operation at this time, there is less likelihood of a severe reaction and a possible osteomyelitis. I am firmy convinced that an external frontal operation is not necessary in mild polypoid involvement, when there is present a small frontal with few, if any, orbital cells, and where, at the time of the intranasal operation, the nasofrontal duct has been sufficiently widened to allow good drainage. I have record of many patients who were completely relieved of symptoms of chronic pansinusitis with this method of intranasal surgical approach to the frontal sinus, plus the removal of severe pathological changes from the antrum. It is right, therefore, to assume that no set operation can be considered in dealing with this problem. The intranasal operation may in some instances be just as complete as an external one.

To know when and how to operate on a patient with chronic pansinusitis is of greatest importance, and is a question which should be given serious consideration by every rhinologist. It is one of the burning questions of nasal sinus surgery, for we must all admit the problem has not been solved. The results of the external ethmofrontal operation will remain problematical because of two unfortunate sequelae—namely, postoperative narrowing of the nasofrontal duct, and the continuation of discharge. Those of you who have performed many of these operations will agree with us that the above statement is correct. Several months following the operation, when the newly formed nasofrontal duct becomes narrowed or completely closed, and retention symptoms make their appearance, the rhinologist is naturally disappointed but has little to say regarding this unfortunate occurrence. Rarely do we find a report of this complication in the literature. As a matter of fact, many papers have been written on the technique of the external ethmofrontal operation, with no mention made of the postoperative closure of the nasofrontal duct. We dislike to speak of our failures.

It is not my purpose to discuss the operative technique of the various sinuses. A few pertinent facts relating to the external ethmofrontal operation, which seems to be the crux of the problem,

may be of some value. My aim, therefore, will be to concentrate attention chiefly on the operative treatment pertaining to the region of the nasofrontal duct. The views advanced here are largely based upon conclusions gathered from a long period of personal observation. During the past few years we have succeeded in keeping the duct patent by paying attention to the following points:

1. A mucous membrane flap of the lateral nasal wall is made.

2. The ascending process of the superior maxilla is removed to almost its entire extent.

3. The lacrimal bone and the anterior portion of the lamina papyracea are removed, preserving the posterior portion of the lamina if possible, so that prolapse of orbital tissue does not follow.

4. A large part of the nasal bone is removed.

5. The mucous membrane of the posterior wall of the nasofrontal duct is left untouched, unless it is found very much involved.

6. The processus frontalis is slowly reduced in size, with a flat chisel, until it is on a level with the medial wall.

7. If the mucous membrane of the anterior inner wall of the frontal sinus is not too thick, a flap is made with the base of the flap medially placed so that it may be carried downward over the remaining portion of the processus frontalis.

8. If the rest of the mucous membrane of the frontal sinus is mildly involved, it is left intact, for we have found that ventilation and good drainage with postoperative irrigation of the cavity with a two per cent calcium chloride solution suffices to bring about the desired result.

When it is found necessary to remove the mucous membrane of the frontal sinus, careful curetting of all recesses is necessary. If temporal prolongations are present the external incision is extended laterally as far as is necessary. Persistence of discharge is to be expected if islands of diseased mucous membrane are allowed to remain. Complete exenteration of the ethmoid cells is important. The posterior laterally displaced ethmoid sinuses, which are most difficult to reach, are cared for with small curved curettes. If the mucous membrane of the sphenoid has undergone hyperplastic changes it is dislodged with small curved elevators. In some cases we have been able to preserve the middle turbinate. A balloon tube is placed in the region of the newly formed nasofrontal duct and is

allowed to remain for two or three days. There is less danger of the flaps being disturbed when this form of dressing is used. This, then, is my present-day point of view relating to the important phases of external ethmofrontal surgery.

In conclusion may I repeat that many cases of chronic pansinusitis do not require a radical operation on all sinuses, and especially is this true if there is a mild involvement in some of the cells. It should be our aim to discover, if possible, which sinus is the chief offender, to eradicate completely the disease therein, and to approach the mildly involved sinus in a less radical manner, if our surgical judgment warrants such a procedure. That complete removal of diseased mucous membrane from all sinuses is many times necessary will not be denied, but that it should be the method of choice in all patients with chronic pansinusitis is, in my opinion, not justifiable. It is almost proverbial that patients who have had several or more nasal or sinus operations, and who continue to suffer with symptoms referable to the sinuses, require radical measures to bring about relief. In common with many other phases of surgical knowledge, external ethmofrontal surgery has had, and is still having, many obstacles to overcome.

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XVII

STUDIES ON BÁRÁNY'S OPERATION ON THE FRONTAL SINUS

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The question of the surgical treatment of frontal sinusitis has been a subject of considerable interest throughout the ages and one regarding which many suggestions have been made. The subject was recently discussed at the Congress in Paris in 1936, in a report of which Aubin and Madura give a very full account of both the clinical symptoms and treatment of this disease.

The operation introduced by Bárány has been employed in Mygind's ward at the Kommunehospital, Copenhagen. In the following paper, I shall give an account of the results obtained, and, by way of comparison, quote those of Bárány[1] himself and of his pupil and close co-operator Soderberg.[2]

TECHNIQUE

There are three groups of operations on the frontal sinus: (1) external, transcutaneous, (2) endonasal, (3) combined forms. Bárány's operation belongs to the first group. The principle of it, as of so many other radical operations on the frontal sinus, is (a) drainage through the natural passages, (b) total ethmoidectomy, (c) plastic treatment of the mucous membrane.

The method of procedure is as follows: After subcutaneous infiltration anesthesia and painting of the nose with cocain, a crescent-shaped incision, from one to two cm. long is made beginning at the medial lower edge of the eyebrow. The edges of the skin can be shifted so that even quite a small incision gives good room. The periosteum is cut through and the lowest part of the frontal sinus, the frontal process of the maxilla, and the nasal bones are laid bare. This is followed by removal first of the floor of the frontal sinus and the very lowest part of the anterior wall (about ½-cm.), then of the frontal process of the maxilla right into the medial orbital margin and almost all of the nasal bone, and finally of the piece of bone still remaining between the two resectional openings, the agger nasi. Bárány originally removed the whole of the lacrimal bone and a large

part of the lamina papyracea of the ethmoid. This procedure has later been abandoned as being superfluous (Soderberg). As in removing the frontal process of the maxilla the anterior wall of the ethmoid has been taken away, the ethmoid can now be completely exenterated, e. g., with Weil's forceps. The bared nasal mucous membrane on the roof of the nasal cavity can now also be seen, and the place where it passes from the lateral wall on to the septum recognized. The diseased mucous membrane of the frontal sinus is removed with a pair of forceps and, finally, an attempt is made by plastic surgery applied to the mucous membrane to encourage the epithelialization of the newly-formed broad opening between the nose and the frontal sinus.

Seiffert's proposal to epithelialize the canal by the free transplantation of epidermis seems, according to the literature, to have found no general acceptance. In theory (a) the mucous membrane of the frontal sinus, and (b) the nasal mucous membrane, are available for plastic surgery. As regards the first, according to Bárány, the wall of the frontal sinus should be chiseled off so that the underlying mucous membrane is not injured. An incision is made in this as shown by the dotted line in Fig. 1. A flap is thus obtained which can be drawn down to cover the medial wall of the newly-formed canal. It has been said that this involves the employment of mucous membrane that is pathological, but from the time when the drainage from the frontal sinus to the nose is re-established the mucous membrane resumes its normal appearance and, therefore, ought not to be removed (Bárány). This opinion was later abandoned, and Bárány removed, as we remove, the mucous membrane of the frontal sinus.

As regards the second, plastic surgery of the nasal mucous membrane can be performed either by taking the mucous membrane entirely from the lateral wall of the anterior part of the nasal cavity or by taking a corresponding flap of mucous membrane from the lateral wall and the septum. If the mucous membrane is taken from the lateral wall of the nasal cavity it can be cut out with the various incisions shown diagramatically in Fig. 2. Should it, on the other hand, be desired to take a combined flap, the plastic surgery of Sourdille should be followed. (Fig. 3.)

Bárány's original proposal was to take a flap from the frontal sinus (Fig. 1) and also one from the lateral wall of the nasal cavity (Fig. 2a), both being used to cover the medial wall of the newly-formed canal between the frontal sinus and the nose. However, he

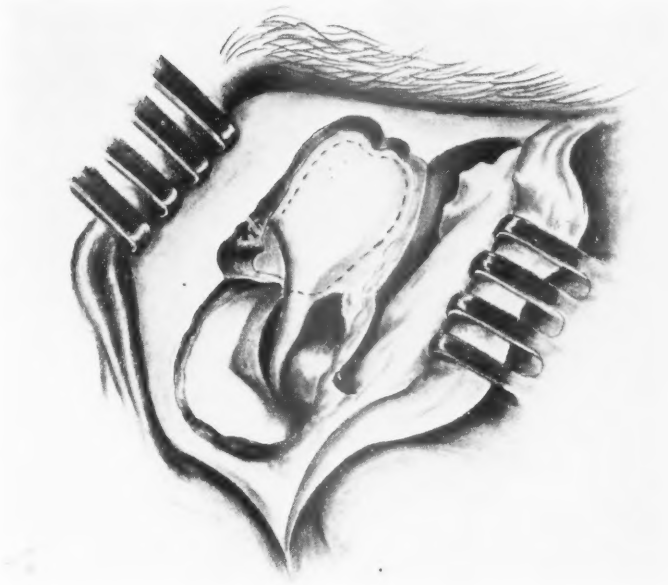

Fig. 1. The mucous membrane in the frontal sinus and in the anterior part of the nose is seen laid bare after the removal of the floor of the frontal sinus (and a little of the anterior wall), the frontal process of the maxilla, and the nasal bone. The dotted line shows the way in which Bárány performed his plastic surgery of the mucous membrane of the frontal sinus.

abandoned his proposal, relinquishing the idea of forming a flap from the mucous membrane of the frontal sinus and performing his plastic surgery by Sourdille's method. The advantage of this large flap is that it is intended to reach right up inside the frontal sinus and thus promote rapid epithelialization; but there is the risk that it may curl up and close the newly-formed canal instead. Sourdille himself has also abandoned this method and performs his plastic surgery as shown diagrammatically in Fig. 2b and Fig. 4.

We have come to the same result quite independently of Sourdille. We used the combined flap in twenty-four cases (Fig. 3), in three of which we had to make a revision, as the flap had curled up

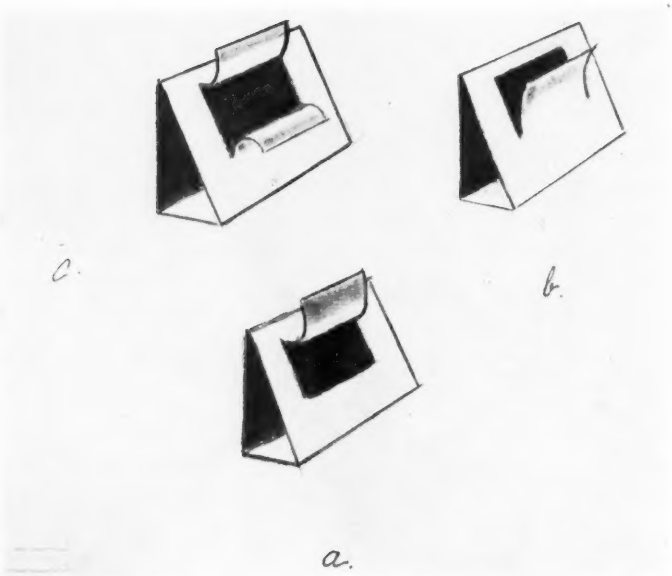

Fig. 2. Diagram showing how the mucous membrane on the lateral wall of the nasal cavity can be used for plastic surgery of the mucous membrane. The drawing indicates the mucous membrane on the septum, the floor and the lateral wall of the nose.

and caused retention in the frontal sinus. In the next twelve cases we, therefore, only formed the flap from the lateral mucous membrane, the result being just as good, with not a single case of these retentions. In six cases the register gives no information as to the method of plastic surgery employed.

A rubber drain is inserted from the nose up into the frontal sinus (and removed on the third or fourth day) and a small cigarette-drain in the angle of the wound. The incision is closed with clips, which are removed two days after the operation.

The material consisted of forty patients, two of whom were operated on bilaterally. Ten of the patients suffered from frontal and ethmoidal sinusitis, and thirty of them from pansinusitis. Eight

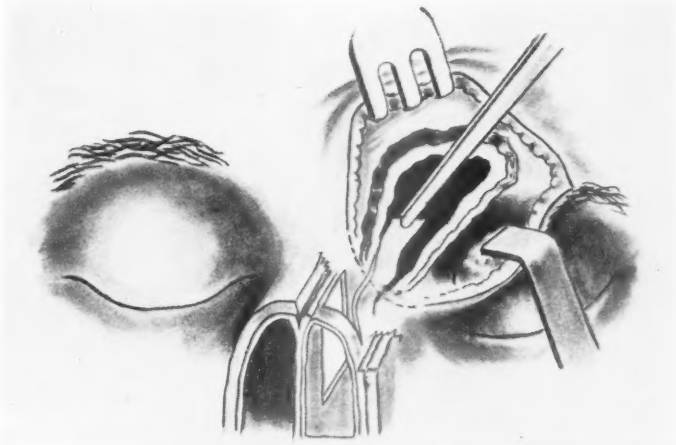

Fig. 3. Sourdille originally formed a corresponding flap of mucous membrane from the septum and the lateral wall of the nasal cavity. He later employed the method of plastic surgery shown in Fig. 2.

had acute suppuration (two with threatening meningitis and six with peri-orbital abscesses); thirty-two were cases of chronic disease.

Re-examination later was carried out in thirty-seven of these cases (one of the last three died some years later of miliary tuberculosis, one is in a mental hospital, and it has not been possible to find the third). Twenty-two were under observation for over three years, twelve for from one to three years, and three for only six months.

A. IMMEDIATE RESULTS

Mortality: None.

Postoperative Complications:

(a) By far the most serious complication was a case of post-operative leptomeningitis (Reg. 175/1933). At the conclusion of the operation, fluid was distinctly seen oozing out from the bottom of the wound (cerebrospinal fluid?) although there was not the slightest suspicion of dural lesion. Resection of the maxillary sinus by Denker's method was performed after Bárány's operation. Men-

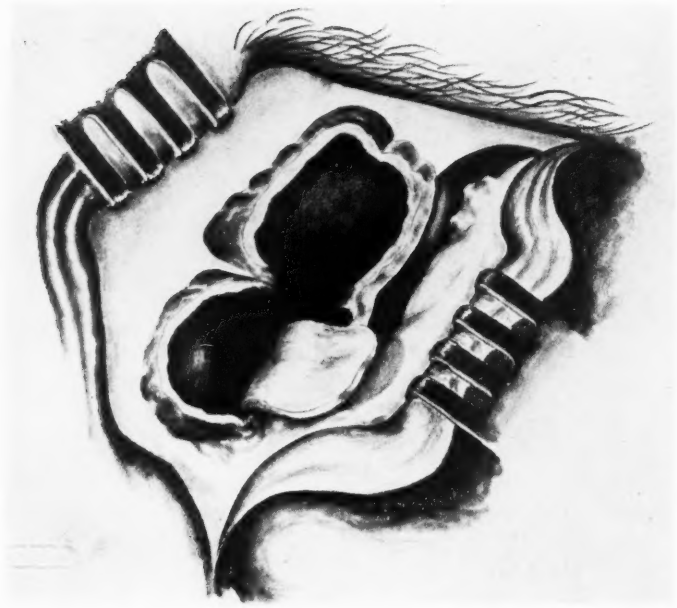

Fig. 4. A short flap of mucous membrane, which can be drawn over laterally, is formed by incision of the nasal mucous membrane on the roof of the nasal cavity.

ingeal symptoms occurred during the course of the day. Lumbar puncture, 5,700/3 cells. Craniotomy was performed through the posterior wall of the frontal sinus extending backwards down through the cribriform plate. The wall of the cranium seemed to be quite intact. The dura looked normal. An incision was made in it. Puncture of the brain gave no result. Painting with iodine. The patient was discharged, cured, twenty days later.

(b) A case of erysipelas.

(c) A case of scarlet fever.

(d) Four cases of diplopia.

All the patients were discharged in good health and received after-treatment, Ritter's bougie being introduced.

B. SUBSEQUENT EXAMINATION

Thirty-six of the forty patients were examined at the hospital clinic, and one elsewhere; three could not be examined for the reasons already given.

I. *Subjective Condition of the Patients After Operation:* As already mentioned, we have information about thirty-seven of our forty patients. The results are as follows:

(a) Twenty-seven of these thirty-seven patients are quite free from all the symptoms caused by their sinusitis. Examination of one hundred and seven of Soderberg's patients was carried out. The twenty acute cases that were operated on were completely satisfactory, both subjectively and objectively. The results of seventy-five of the eighty-seven patients with chronic sinusitis were also quite good. The remaining twelve were all better subjectively, but not entirely so.

(b) Four patients complained of either slight periodical discharge from the nose or occasional pain in the region of the frontal sinus operated on. This occurred especially when they had colds. The frontal sinus operated on might thus be thought to be a locus minoris resistentiae. Ritter's bougie slid up easily in all the cases, and there was no secretion to be washed out. All in all, these patients were satisfied with the results of the operation; the symptoms that remained being quite inconsiderable.

(c) Finally, there were six patients that still complained of pains, discharge and headache. These were five women, two of whom were very nervous, the sort that suffer periodically from a "nervous breakdown," and one man. The following explanation probably accounts for the non-disappearance of the symptoms: One had empyema of the maxillary sinus on the side not operated on; in the case of the second, the maxillary sinus operated on had closed again; the symptoms due to the third's frontal sinusitis (secretion of pus, frontal headache, pus in the frontal sinus operated on) had disappeared completely, but the patient had also had vasomotor rhinitis before the operation, and symptoms of this (sneezing, very watery, but never purulent, secretion) were still present.

There thus remain three of these unsatisfactory patients: One recovered after a short treatment consisting of irrigation of the sinus that had been operated on; no explanation could be found for the bad result in the second case. The bougie went up easily and there was no secretion in the frontal sinus.

The result in the case of the last of these three patients was very bad. This was a 28-year-old woman who suffered from bronchial asthma. She had undergone removals of polypi from both sides of the nose for many years, but they constantly grew again. Both maxillary sinuses were first operated on, but, strangely enough, no further polypi were found, though there was a secretion resembling chewing gum. A year later her left frontal sinus was operated on; this was full of polypi. Her left maxillary sinus was opened again and this time contained a great many polypi, in spite of there being a large broad opening to the nose. We did not see her later, but she has been treated elsewhere for asthma, and we have been informed that both sides of the nose were again chock-full of polypi, in both posterior nasal orifices and in the newly-formed canal to the frontal sinus. There was a small fistula in the cicatrix from which a little serous secretion could be pressed.

This result was, thus, very bad. The operation ought certainly never to have been performed; it should have been enough just to keep the nasal passage free by periodically removing some of the polypi. Soderberg has two similar cases in his material. He is also of the opinion that such cases are not suitable for operation.

II. *The Objective Result:* (a) The appearance of the patients after operations. The complaint has been made, especially formerly, against the external transcutaneous methods that in so many cases they resulted in disfiguration of the contours of the face. This was so especially in the operations in which the anterior wall of the frontal sinus was sacrificed so that the cutis and the subcutaneous layers subsequently fell in at the spot. This led, as is well known, to Killian's publication of his operation, which preserved a span to prevent the cutis and the subcutaneous layers from falling in later. In Bárány's operation, however, only a part of the floor of the frontal sinus and an inconsiderable part of the anterior wall are removed, and we have not seen a single case of disfiguration in the form of falling in of the cutis and the subcutaneous layers.

Cicatrices: We have seen thirty-six patients at the clinic who had been operated on (two double operations). Of these thirty-eight cicatrices, twenty-six were ideal, i. e., they were almost invisible in most cases. It was often necessary to look very carefully to discover that the patient had been operated on.

The cicatrices were good in five cases, by which is to be understood that they were completely level with the rest of the skin and not discolored, but yet were visible as a stripe.

There were seven bad cases, i. e., the cicatrix either stood out like a seal, or was a little deeper than its surroundings, or was broad and visible. It is very easy to show the cause of these bad results. All the cases are those of the results of suppuration which either was present at the time of the operation (peri-orbital abscess) or made its appearance during the operation, necessitating drainage for a longer or shorter period.

In other words, there is no question of disfiguration after Bárány's operation. The wound is, to all intents and purposes, invisible in most cases if it heals aseptically.

(b) *The Passage to the Nose:* The question is, whether the new connection formed by the operation between the frontal sinus and the nose is still open or has closed again. To deal with this we introduced Ritter's No. 4 bougie. Two of the patients were nervous and did not want to have it introduced. Both were, however, quite well, and the former great discharge from the nose and pain in the frontal sinus had quite disappeared. It is permissible to conclude from this that the passage was clear.

Thirty-four patients remain (two with double operations). Ritter's bougie went up with the greatest ease in thirty-four cases, the largest (No. 4) thirty times and No. 3 four times. In one case considerable deviation of the septum to the same side prevented its introduction. In one case attempts to introduce the bougie were unsuccessful; no explanation was found for this. This patient, too, was in good health.

In other words, the operation had served its purpose so far as this was to form a constant broad connection between the frontal sinus and the nose.

(c) *Ocular Complications:* For a particular reason I have chosen to delay the discussion of ocular complications until now. There were four patients in this group. Were they to be asked, "Did you have double vision after the operation?" three of them would reply "For a short time after the operation but not now." If they were examined by an oculist it would appear that three of them were suffering from paresis of the oblique superior, and the fourth from some tightening of the cicatrix in the orbit. But their double vision does not trouble them at all and is only noticeable when looking downwards to the greatest extent and to the side not operated on. Only one patient reported spontaneous double sight when looking downwards.

The action of the oblique superior is, as is well known, to lower the eyeball and to rotate it inwards. Paralysis will therefore be evident by a double image on the horizontal plane, especially when the patient is going downstairs, looks downwards, as when reading, etc.

Paresis of the oblique superior is one of the most frequent pareses of the muscles of the eye. These can be divided into two groups: (1) those due to paralysis of the n. trochlearis, and (2) traumatic, due to the loosening of the trochlea by an operation. Our cases belong to the latter, by far the smallest group.

The consequence may be temporary diplopia. Even if the trochlea does not grow fixed firmly in exactly the same place, the patient will involuntarily suppress the double image in the course of some time, though thorough examination will, nevertheless, reveal the paresis. If, however, the displacement is great, the patient will have permanent diplopia, which can be very annoying and necessitate Halle's operation by which the periorbit is drawn into place by means of a mattress suture through the periorbit and the periosteum in the superior medial corner of the eye. We are, therefore, extremely careful in replacing the periorbit.

It may be said without any doubt that our three cases had a traumatic etiology of this sort. In one case, that of the patient with postoperative leptomeningitis, it was necessary to perform major craniotomy through the anterior and posterior walls of the frontal sinus and the cribriform plate. In the second there was a large rupture in the periorbit, and, later, severe edema of the eye, with protrusion and dislocation outwards and downwards, which disappeared later. The course of the third case cannot be read so easily from the history, but there is hardly any doubt that the etiology is the same.

Our last patient belongs to the second group of diplopia. He had quite an uncharacteristic double image when looking to the side not operated on. The oculist is of the opinion that this is due to tension of the cicatrix. It appears from the history that the operation took a long time and was difficult and that there was a great deal of bleeding. It might be thought that some tension occurred by the organization of bleeding between the periorbit and the bone, and that this prevented the full movement of the eye.

Soderberg has three cases of diplopia in his material; since taking particular care in the postoperative fixation of the trochlea he has had no case of diplopia.

Frenckner[3] had one patient, out of forty-one cases, who was troubled with diplopia, and two more in whom, although they were not troubled with it, diplopia could be ascertained when they looked in certain directions.

This emphasizes the great importance of extreme care when replacing the periorbit, and the value of as exact fixation of the trochlea as possible if it should be loosened during the operation.

I wish to thank my colleague Dr. V. Gestsson heartily for the drawings he has made for this paper.

Ostergade 18.

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XVIII

SOME PRELIMINARY EXPERIMENTS IN THE STUDY OF CIGARETTE SMOKE AND ITS EFFECTS UPON THE RESPIRATORY TRACT*†

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SAINT LOUIS

I

BACKGROUND

Perhaps one should not allow a work so purely preparatory as this one to find its way into print. Extenuating circumstances sometimes exist, however, which justify the preliminary sketch of a problem such as this one.

First, the effect of cigarette smoke is a subject of great general interest. Second, smoke is a substance so evanescent that it can scarcely be approached as a substance at all, and so complex that it is likely to require years of study before any definitive report of its effect on the individual will be available. And third, so much unsubstantiated opinion is bandied about in regard to it that laryngologists may conceivably overlook the immaturity of this report on the ground that a little information is better than none at all.

At least, one has been at some pains to devise methods which will ultimately yield dependable and accurate information. More will have to be found before any valid conclusions can be drawn.

The study of cigarette smoke and its effect upon the respiratory tract presents a fascinating problem to the investigator for a number of reasons. In spite of the general interest which it arouses, surprisingly little scientific research has been recorded. It is not without some trepidation that one undertakes to investigate a matter which has been the subject of so much pseudoscientific comment, for it is difficult to evaluate what has gone before. Some of it has been unbiased. Much more, however sincere, is colored by prejudice

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which, we are told, has surrounded the habit of smoking since Sir Walter first introduced it to the Old World.

For this reason it was determined to reverse the usual process, to remain deaf and blind to all previous writings and conceptions, and to begin by setting up the machinery for uncovering some cold facts as competently as it lay in one's power to do. Much information so received is, of course, already well known. No matter. It comes first-hand, and serves as a reliable background for our future efforts.

As a beginning, all manner of heterogeneous data were assembled, both as to the habits of smokers and to the characteristics of cigarettes, cigarette tobacco and cigarette smoke. Some of these, which have a direct bearing upon the details of the investigation, are enumerated.

The Cigarette:

The typical American cigarette is 7 cm. long, .9 cm. in diameter, and weighs from 1 gm. to 1.1 gm., depending upon humidity, packing and a number of other factors.

It consists chiefly of American type tobaccos, with an admixture of Turkish, Egyptian, Greek and others, cured according to various processes.

The tobacco is treated with some hygroscopic agent which causes it to hold the desired amount of moisture to permit handling, to burn properly, and to retain its pliability until it is smoked.

It is flavored with mixtures of volatile substances which impart to the individual brands their characteristic tastes and aromas. Although these are generally known, the exact formulas in each case are carefully guarded secrets.

Great uniformity of the product is maintained in modern cigarette making.

The Smoke:

The chemists tell us that the combustion products of the burning tobacco leaves which constitute smoke are present in almost unlimited variety, and depend not only upon the character of the burning material, but also upon the circumstances attending the combustion, such as:

The position of the cigarette while burning,

The speed of the puff,

The volume of the puff,
The frequency of the puffs,
The number of the puff in the series,
The humidity of the tobacco and of the room.

It quickly becomes apparent that if the contemplated experiments are to have any value, some means must be employed to produce a smoke of fairly constant characteristics; and further that these characteristics must correspond in some way to those at work upon the mucosa of the average smoker, if indeed such a one exists.

To this end it was arranged to study the habits of cigarette smokers unobserved. With the aid of a stop-watch this was done hundreds of times. The results were so diverse that a mean rather than an average was finally determined upon.

The interval between puffs was found to vary from fifteen seconds to more than a minute. This seems to depend partly upon habit, and partly upon the incidental occupation of the individual, such as reading, conversing, eating (!) or driving.

Some smokers discard each cigarette after a few puffs; most, however, discard them when they become too hot to hold.

Some inhale deeply, some not at all.

Those who blow the smoke through their noses are apt to do so habitually.

Experiments upon attaches of the Institute (without telling them the purpose of the test) determined the volume of the average puff to be between 25 cc. and 40 cc. Singularly, some of the higher volumes were recorded among the women.

The total volume of respiration involved was usually around 500 cc.

It was decided from the foregoing, that the smoke mixture to be investigated should be that of a mouthful of smoke from the cigarette, diluted with a lungful of air. In actual smoking, this is the mixture which comes in contact with the mucosa of the nose or of the trachea, depending upon the direction of flow. When a subject inhales, he gradually mixes his mouthful of smoke with the inspired air as it passes from his nose through his pharynx. This mixture passes into the trachea. Or, if he blows smoke through his nose, again he mixes his mouthful of smoke with a lungful of air in the pharynx, only this time the mixture acts upon the nasal mucosa.

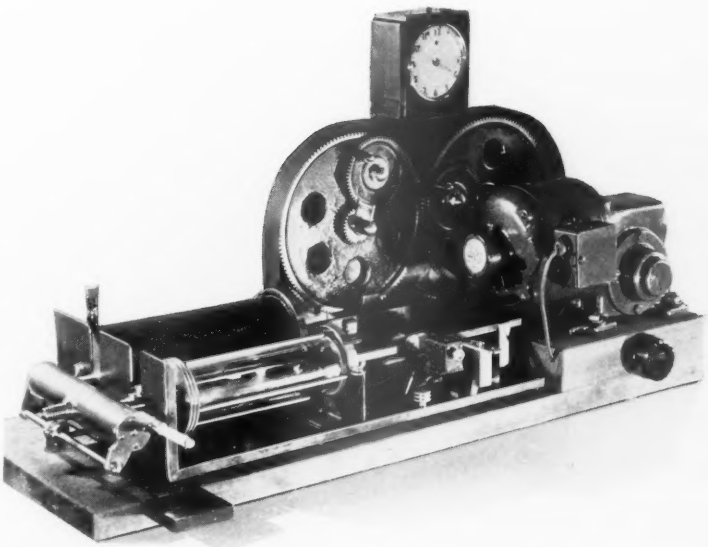


Fig. 1. Smoking machine. This device takes a 35 cc. puff from the cigarette at the left, every 30 seconds, dilutes it with 500 cc. of air and delivers it for use in experiments.

II

THE SMOKING MACHINE

To build an apparatus which would draw a fixed amount of smoke from a cigarette, at uniform intervals, and deliver it mixed with a definite amount of air, seemed a fairly simple task. It did not prove to be so. Four such machines had to be built before the result was satisfactory.

The difficulty lay always in the failure of the machine to perform uniformly after a certain amount of tar had been deposited within it.

All rubber had to be eliminated.

All valves which depended upon positive or negative pressures within the machine had to be eliminated, as their operation was im-

peded by the tar, and with the change in their capacities the smoke mixture was altered.

For the same reason, everything adjustable had to be replaced by something not adjustable, since it could not be certain that the adjustments would remain unchanged over the period of months or years required for the experiments. The exact concentration and combination of the product is less important than that it shall remain uniform.

In short, a machine had to be constructed entirely of metal and glass, whose pistons, timers and valves are all activated accurately through a chain of gears, by a motor of sufficient power to remain unaffected by changes in temperature, lubrication, tar deposit or other variable, including fluctuations in the line voltage. (Fig. 1.)

The present device, which has functioned perfectly for a year or more, takes a draw of 35 cc. from a cigarette at intervals of thirty seconds. This it dilutes with 500 cc. of air and expels through a tube. The cycle occupies exactly thirty-six seconds.

All passages are of ample diameter, and are streamlined, including those in the interior of the mechanical valves, in order to reduce eddies, and hence tar deposits, to a minimum.

Humidity readings are taken, not in the machine itself, but in the room in which the experiments are performed.

A mechanical device records the number of puffs so that these shall not vary when comparative results are required.

The diluted smoke passes through a large short tube to a metal box in which the experimental animal, usually a rabbit, is closely confined. A tube, connected to the vacuum system of the laboratory, clears the air in the box of smoke so that the animal breathes fresh air between puffs.

At first the rabbits attempt to avoid breathing while the smoke is passing, but they soon become accustomed to it, and breathe regularly.

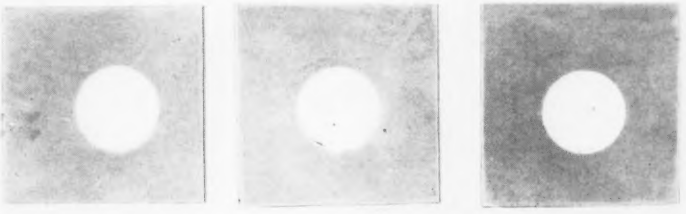
III.

SOME PHYSICAL QUALITIES OF CIGARETTE SMOKE

One of the fundamental problems is the actual physical behavior of cigarette smoke in passing through the chambers and passages of the respiratory tract.

As everyone knows, these airways are tortuous and complicated. Sometimes round, but more often triangular or even slitlike in sec-

Fig. 2. Smoke from the tenth puff has been allowed to settle on white glazed paper. The square on the left shows the deposit after 5 minutes, that in the center after 7½ minutes, that on the right after 10 minutes. (Tobacco mixture B.)



14th puff 1st puff 20th puff

Fig. 3. Settling time: 15 minutes. (Tobacco mixture A.)

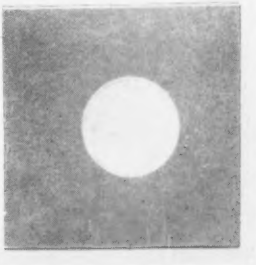


Fig. 4. One puff from the end of a cigarette burned without being smoked. (The region of the cigarette corresponding to the 20th puff.) Settling time 15 min. (Tobacco mixture B.)

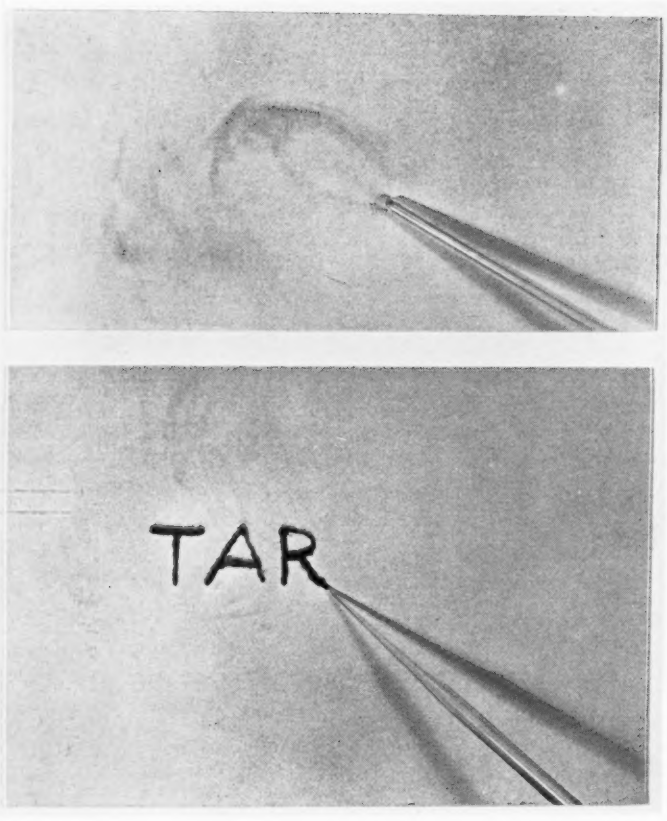


Fig. 5. The upper figure shows the smoke being blown through a glass tube onto a white sheet of paper. A single puff leaves no visible stain.

Fig. 6. The lower figure shows the dense deposit of brown tar which results from blowing the smoke through the same tube drawn to a fine point.

tion, now large, now small, now vertical, now horizontal, they present a series of tunnels and baffles to the smoke as it passes through them.

Studies were made to determine what effect, if any, these characteristics might have upon the impact and consequent deposit of tar upon the mucous membrane. The first of these consisted in passing the smoke at various rates and pressures through glassware of many types and shapes and observing the effects.

Certain fundamental phenomena were noted which have an important bearing upon the general problem. These phenomena are familiar to all heating engineers, but in the light of their present application it will repay us to consider them.

Smoke from a cigarette consists of tarry substances which occur as minute globules, together with the distillates of certain volatile substances which are vaporized by the heat before the actual combustion point is reached. There is also, of course, some water vapor. The tarry globules seen under high magnification are infinitesimal specks suspended in the air.

Smoke is heavier than air and if there are no currents mixes very slowly with it.

Experiment. Smoke conducted to the floor of a large Erlenmeyer flask, for example, lies there in a dense stratum. If the flask is tilted, the surface remains horizontal and is little disturbed. The smoke can actually be poured from the flask like any liquid; if the flask is righted before all the smoke is poured out, the remainder gravitates to its original position on the floor.

This suspension continues for long periods. In small, tightly closed chambers, it is a matter of minutes before the globules settle to the bottom to be deposited as a tarry stain.

Experiment. A small box was prepared with a sliding lid so as to be airtight to the extent of excluding air currents in the room. A small glass tube was sealed into a hole at one end. The floor was covered with a square of glazed white paper. A mouthful of cigarette smoke was now blown into the box through the glass tube, and the box was set in a protected place, in order to permit the smoke to settle undisturbed. A coin was laid on the center of the white paper to protect it from the tar and furnish a white spot for comparison.

With this simple device it was found that at least five minutes was required for the smoke to settle sufficiently to discolor the paper at all. The stain deepened more rapidly in the seventh and eighth minutes, but it usually required nine or ten minutes to collect all the smoke. (Fig. 2.)

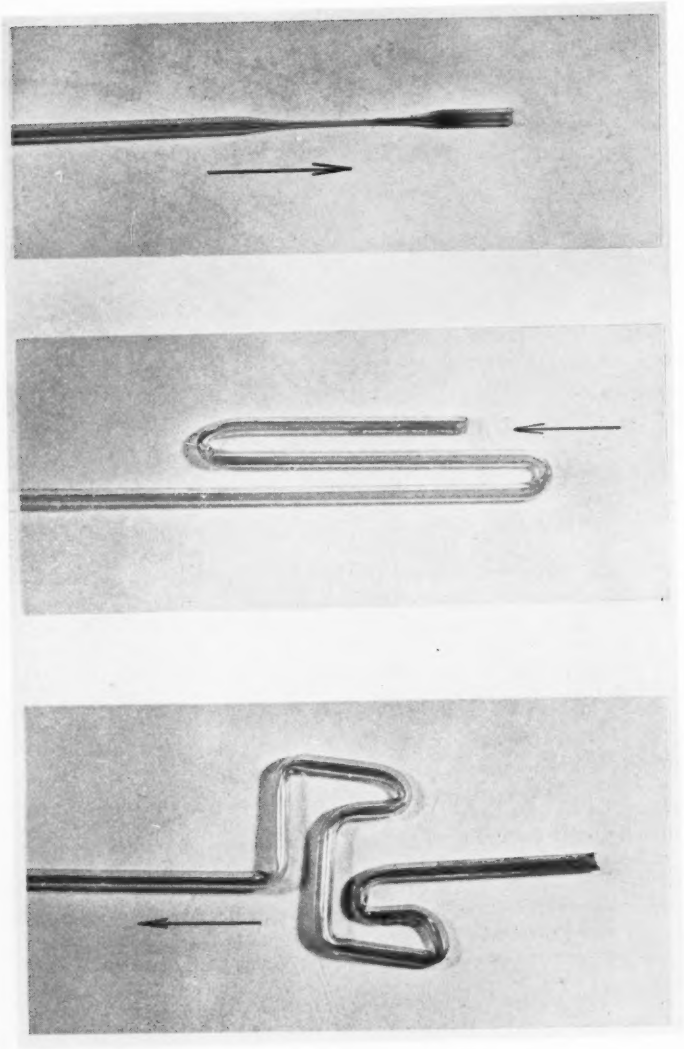


Fig. 7. When smoke is blown through a tube in which there is a constriction the tar is deposited at the distal end of the constriction where the eddies which occur drive it against the walls.

Fig. 8. Smoke passed through a tube in which sharp bends occur is deposited as tar at the bends; more densely at the distal end, again where the globules are driven against the walls. This occurs only when the smoke is passed briskly as in blowing it from the mouth or nose.

Fig. 9. When the smoke is passed slowly through the tube—at the speed of drawing it through the cigarette, the deposit forms more slowly, and is more generally distributed.

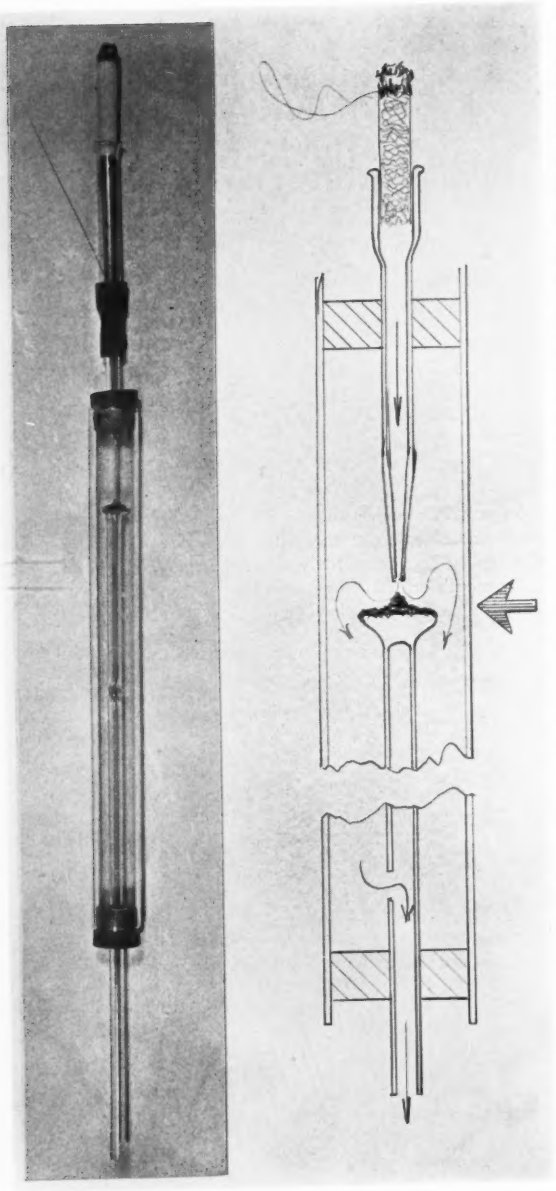


Fig. 10. Mechanical tar filter. When the smoke from the cigarette at the right is drawn through the capillary tip of the tube into which it is inserted, and projected against the glass disc (indicated by the large arrow) tar piles up on the disc very rapidly, and almost no smoke issues from the left end.

It will be seen therefore that surfaces in casual contact with smoke collect very little of the tar. There are important exceptions, however, as is shown by the following:

Experiment. Smoke from a cigarette is blown through a short length of glass tubing (¼-inch diameter) against a clean white paper surface. This can be done many times without the paper or the tubing showing any stain. (Fig. 5.)

If the tip of the tube is drawn out to resemble the point of a lead pencil, so that the orifice is only a millimeter or so in diameter and smoke is again blown through it, a dense brown streak is left on the paper. (Fig. 6.)

In the first case, the tar globules pass through the tube in parallel courses, and fail to touch the paper as the air stream carrying them is gently deflected.

In the second case, the globules passing through the tube are suddenly brought in contact with one another, coalesce and are sent in sharp eddies against the paper, to which they adhere.

Smoke blown through the unconstricted tube under increased pressure leaves a slight stain on the paper.

Smoke blown through a tube of similar size but kinked and twisted upon itself, is deposited upon the walls of the tube at the distal part of each kink; in other words, where eddies occur. (Fig. 8.)

Smoke blown through a tube in which a constriction has been made is condensed and deposited at the distal end of the constriction, and for some distance beyond, again where eddies occur. (Fig. 7.)

This is the principle involved in removing some of the tar by smoking one cigarette through another. The second cigarette merely constitutes a series of capillary spaces through which the smoke is forced to pass. Some of the tar is left behind, and some of the distillates condensed. Precisely the same result can be obtained by smoking only half a cigarette, or by smoking one of half the usual cross section.

Whether the tar which constitutes the visible constituent of the smoke contributes more than a visual esthetic effect is a moot question. In large quantities it would certainly be unpleasant. In the minute quantities which reach the tastebuds it probably contributes to the enjoyment of the smoke.

The pleasurable constituents of cigarette smoke are largely condensable. Seven cigarettes were attached together, tandem, with cellulose tape, making one long cigarette 19¼ inches long, and without

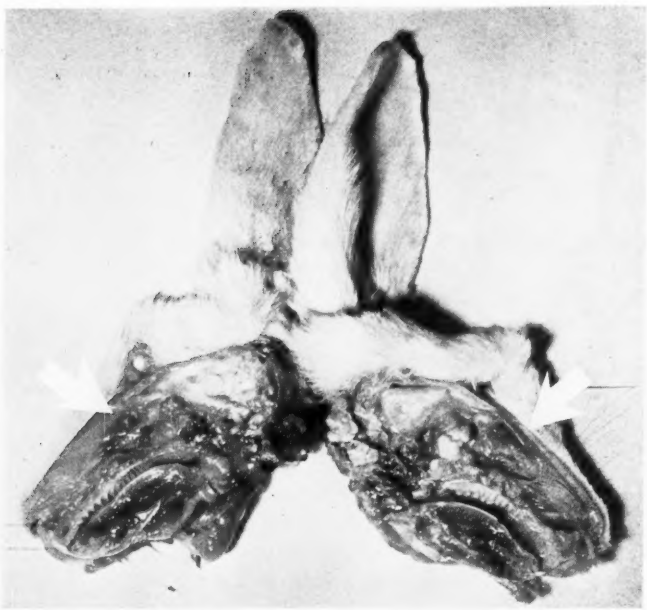


Fig. 11. Dissection of the nose of a rabbit which has inhaled the smoke from a dozen cigarettes as delivered at 30-second intervals by the smoke machine in the course of one day. The arrows indicate the brown deposit on the septum, left, and the lateral wall, right.

leaks. Although this was smoked hard so that the lighted cigarette at the tip burned furiously, what was blown from the lips was scarcely visible, and practically without taste.

Second, a purely mechanical device was constructed which produced the same effect even more simply. This was made of glass and demonstrated exactly what occurred. (Fig. 10.)

Experiment. Inside a large glass tube, a smaller glass tube with a capillary tip was fixed in such a position that when smoke was drawn through the device it was projected from the capillary opening against a flat glass surface. The result was that practically all the tar was piled in a heap upon the glass surface in incredible amounts. The smoker's reward at the other end was scarcely worth the match.

The discussion of the nicotine content has been purposely omitted from this discussion, as it constitutes a separate problem.

The foregoing observations lead to certain deductions, which at the present status of the work must not be admitted as conclusive, but for which further experimental evidence already exists.

It is seen, for example, that smoke held in the mouth or the nose, or even the trachea, for a second or two and then expelled, leaves behind it in those cavities only the most infinitesimal amount of tar. One must not fall into the error of assuming that other effects, as for instance, vagus stimulation, must be equally slight. Since, however, it requires minutes for smoke to settle in quiet chambers, and since the tar globules rarely adhere to the walls of straight and unobstructed tubes, little remains of the smoke itself. (A large straight rubber tube which has been in service for months in the smoke machine described shows scarcely any discoloration on its inner surface.)

The airways of the mouth, nose and throat are often not straight, and not unobstructed, and in such locations tar deposits occur. The lingual surfaces of the upper incisors, the tip of the tongue where eddies occur when smoke is drawn in; often show discoloration. The probability that constrictions in the nose caused by deviations and spurs would cause accumulations of tar, suggested more experiments, this time on rabbits.

Some months ago, while the sinus of a living rabbit was being observed under the microscope for other purposes, cigarette smoke was blown into it through a capillary tube, with the intention of watching its possible effect on the cilia. The dark brown spot which appeared indicated the experiments with the glass tubes just described, and also the following:

Experiment. Rabbits were placed in the smoke machine and made to smoke from ten to twenty cigarettes in the course of a day. At the end of the day the rabbits were killed, their heads split asunder in the midline and the mucosa inspected for deposits of tar. (Fig. 11.)

The tar deposits were not generally distributed, but were confined to the regions of the upper turbinates and the septum opposite. None was found immediately within the vestibule. The nostril of the rabbit is wide and the most constricted portion of the passage lies just anterior to the stained area. Based upon the observations of the constricted tubes mentioned above, the stain might be expected to occur just where it was found.

The stains corresponded so closely to the olfactory regions which are pigmented, that they were first thought to be pigment. However, microscopic examination showed the brown deposit to be on the surface. Furthermore, similar brown stains were produced at will by blowing smoke elsewhere upon the membrane through capillary pipettes.

The deposit of tar so produced stopped the action of the cilia, probably by its mechanical presence, and by the drying action of the smoke stream. This occurred only when the smoke was blown through the pipette, producing the stain. Smoke projected through unconstricted glass tubes in such a manner that no stain was produced had no apparent effect upon the cilia or their behavior.

IV.

AN INQUIRY INTO THE RELIABILITY OF SIMPLE COMPARATIVE CLINICAL OBSERVATIONS OF THE PHARYNGEAL MUCOSA

Since this study must sooner or later concern itself with the effects of smoke upon the clinical status of the human membrane, some means will have to be found, and some standard established against which clinical changes in the membrane can be charted.

By the very nature of the observation the human element must creep into the equation. Minor changes in color, shade and hue must not only be noted, but must conform to some standard. Subtle differences in the behavior of the lymphatic elements of the pharynx, for example, may prove important. In all likelihood even essential changes will prove to be slight.

If day-to-day comparisons are difficult, what credence shall be given to month-to-month observations, especially when these are made by several persons?

It was thought essential to put such observations to the test, to satisfy ourselves at first-hand whether, conducted under ideal conditions and careful control, they could be sufficiently reliable to serve as a basis for any responsible opinion.

It was done in this way:

Survey. Eight observers were chosen from among the Clinic staff. All were young men, all were trained in this clinic, and all had had at least three years' service in the clinic.

Twenty-six subjects were observed over a period of five weeks. Sixteen were medical students, ten were secretaries, assistants and nurses in the clinic.

Neither the observers nor the subjects knew the purpose of the tests and carried them out blindly according to our schedule. Nor did they compare notes. The findings were dictated to an assistant who kept the records.

The observers were instructed to examine the throats of the subjects and to classify their findings according to the following:

Class 1. Throats having no suspicion of any inflammatory reaction. Throats which one could without the shadow of a doubt regard as "normal."

Class 2. Throats showing minor signs of irritation or inflammation of no clinical importance, but merely sufficiently affected to be excluded from Class 1.

Class 3. Throats definitely red and inflamed.

Class 4. Severe sore throats, of the type usually associated with fever and other systemic symptoms. (Only one of these was actually encountered.)

The classification was purposely made so broad that there could be no possible misunderstanding in this regard on the part of the examiners.

All tests were made at one time of day (3 p. m.) and as quickly as the subjects could be moved about, in order to prevent their drinking water or otherwise upsetting their throats between observations. The observers were cautioned to use gentleness in making their examinations so as not to set up any irritation in the pharynges. That this was done is shown by the fact that progressively increasing redness in individual throats was scarcely ever recorded.

One thousand two hundred and ninety-three observations were made.

There were 280 subject-days. (That is, the number of subjects, multiplied by the number of days each was examined by the eight observers.)

The figures were first broken down to determine the agreement or disagreement which existed among the eight observers.

On the 280 subject-days,

All observers agreed	46	times, or	16.4%
There were 2 opinions	188	times, or	67.1%
There were 3 opinions	32	times, or	11.4%
There were 4 opinions	1	time, or	.4%
Incomplete*	13	or	4.6%

*Those marked incomplete were for one reason or another not examined by four or more examiners.

In short, there was some diversity of opinion in 78.9%, notwithstanding the broad division and the concise definition of the four classes.

In each case the subject was asked whether he was aware of any soreness or discomfort or irritation of his throat, in order to compare his subjective feelings with the clinical findings. Naturally the examiners were not informed of his statement.

Of the 280 subject-days, there were only 22 on which the patient was aware of a sore throat.

Of these 22,

8 were recorded as normal by 1 observer — 8 discrepancies
3 were recorded as normal by 2 observers— 6 discrepancies
6 were recorded as normal by 3 observers—18 discrepancies
1 was recorded as normal by 4 observers— 4 discrepancies
1 was recorded as normal by 6 observers— 6 discrepancies

Total 42 discrepancies

Only one was not recorded as normal by any of the observers.

Conversely, when the subject was unaware of any irritation,

in 606 instances he was classified as 2 (negligible redness)
in 46 instances he was classified as 3 (sore throat)
in 1 instance he was classified as 4 (extremely sore throat)

653
42 (from the preceding paragraph)

695 discrepancies (in 1293 observations) between subjective and objective symptoms, or 53.7%.

It is readily admitted that persons exhibiting the slight redness classified as 2 would probably be unaware of any irritation. Revising our percentage, therefore, on the basis of those unaware of any irritation, who were nevertheless classified as 3 or 4, which amounts to dividing our subjects into two broad classes instead of four, we have a total of 89 in 1293, or 6.88 per cent. Such a broad division would of course be useless for our present purpose.

The records were carefully studied to find out whether any of the observers either through personal idiosyncrasy, misunderstanding, faulty illumination or even possible color-blindness might be consistently in error. No such tendency was discovered. No observer was found to be consistently high or low in his grading.

Neither space nor the reader's patience justifies the publication of the protocols here; however, certain collateral data should be included, as they bear directly upon the validity of the results.

First, the observations were made between April 5th and May 9th, 1938. Each day at the hour of the examination were noted the state of the weather, the temperature and the relative humidity. Three days were rainy, two were cloudy and the rest were clear. The temperature ranged from 54 degrees Fahrenheit to 84 degrees Fahrenheit. The relative humidity ranged from 33 per cent to 64 per cent.

No parallel could be detected between the state of the weather and the percentage of Class 3 and Class 4 throats reported. The largest number of red throats (11.12%) occurred on April 12th, a clear day with a temperature of 82 degrees and a relative humidity of .36. The smallest number (1.32%) occurred two days later, when the temperature was 80 degrees and the humidity .47.

No epidemic of upper respiratory infection prevailed during the time covered by these records.

It should be added that the subjects were chosen without reference to their smoking habits, as the observations were intended purely to determine the reliability of human observation of throats at large.

It developed that eleven of the group were cigarette smokers. One was an occasional smoker, six were non-smokers.

Under this heading the following figures are of interest so far as they go:

	Class 1	Class 2	Class 3
4 Smokers	77	140	17
4 Non-smokers	77	109	11

The information contained in the last few paragraphs should be given no more weight than it deserves. It is incidental to the principal experiment which was controlled for the purpose for which it was intended.

To summarize: Regarding the reliability of simple clinical observation as a basis for statistical studies of the pharynx, even under the best conditions and with the problem clearly defined in the mind of the examiner, we find a diversity of opinion reaching 79 per cent.

Regarding the comparison of subjective irritation with objective changes, as seen in the clinic, we find a discrepancy in 54 per cent of the cases, when these are divided into four broad categories. Even when they are divided into two classes, "obviously inflamed" and "obviously not inflamed," the discrepancy still reaches approximately 7 per cent.

Just what are the objective changes produced in the mucosæ by the protracted action of cigarette smoke has not been determined. It is not at all certain that they may be classified in terms of color, or even that progressive irritation results in progressively heightened color.

It would of course be desirable, in the light of the minute changes which are likely to occur, to eliminate the human element as much as possible in recording them.

Whether a colorimeter can be devised which will provide a graphic record of small, variously colored areas of the mucosa and whether after that, one can learn to interpret such a record in terms of cigarette-smoke-effect remains to be seen.

The actual investigation has not begun. We are still making—and discarding—tools.

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XIX

ACUTE ACTINOMYCES MASTOIDITIS*

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AND

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Since the original observations of Israel,[1] Bostroem[2] and others on actinomyces, there has been a profusion of experimental and clinical reports on the genus actinomyces and actinomycotic infections in animal and man. Yet Sanford and Voelker[3] in 1925 were able to collect only 670 reported cases of human actinomycosis in the United States. While the true incidence of human actinomycosis in the United States is undoubtedly higher than appears because of unreported and undiagnosed cases, actinomycosis in man remains an infrequent, though no longer rare, infection.

Statistical survey of reported cases reveals that the actinomyces may attack any organ, with predilection for the cervico-facial, abdominal and thoracic structures, accounting for 60, 18 and 14 per cent, respectively, of the infections in man.[3]

Among the rarer distributions are those which the otolaryngologist is apt to encounter, such as acute and chronic otitis media,[4] granuloma of the nose,[5] sinusitis,[6] tonsillitis[7] and mastoiditis.[4]

It is the purpose of this paper to report a case of acute actinomyces mastoiditis, to review the literature on this subject and to discuss its pathogenesis.

REPORT OF A CASE

CASE 1.—P. V. M., Jr., a 10-year-old boy of Italian extraction, was admitted on February 8, 1937, at the Hospital for Joint Diseases to the service of Dr. J. A. Haiman with the complaint of a discharging left ear, pain in the left mastoid region, swelling of the left eyelid and left side of the face, high fever and severe malaise.

The boy's history revealed that some five weeks before admission he began to experience pain in the left ear; several days later spontaneous rupture of the left ear drum occurred with moderate purulent discharge. Intermittent otorrhea, unaccompanied by any constitutional complaints, continued until two days before admission when the immediate complaints, aforementioned, appeared. There was

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no history of tooth extraction, mouth infection or trauma. No culture of the aural discharge was taken. The patient's past and family histories were negative.

Physical examination on admission revealed a toxic boy with a temperature of 105 degrees. Slight swelling of the left side of the face, moderate edema of both eyelids, ptosis and definite lagging of the left upper lid were present. No exophthalmos was observed; extraocular movements were normal. A thick, yellowish green discharge filled the external auditory canal, the roof of which was sagging; the left ear drum was grayish, thickened and presented a small perforation anteriorly. The left mastoid region was tender and slightly edematous; no fluctuation could be detected. Examination of the right ear, nose, throat, heart, lungs and abdomen was negative.

The clinical impression was one of chronic otitis media and acute suppurative mastoiditis; thrombosis of the left cavernous sinus was feared because of the eye findings.

A simple left mastoidectomy was performed about one hour after admission. The operative note reads: "The external plate of the left mastoid process was removed with the immediate escape of a small amount of yellowish pus and granulation tissue. A culture was taken of this. With alternate use of the curette and bone forceps, the necrotic bone and granulation tissue were removed. Complete exposure of the sinus plate and exploration of the zygomatic cells, which were found to be involved, was performed. The wound was then packed with iodoform gauze."

Postoperative course: On the day following operation, the child was very toxic and somnolent. A complete palsy of the left external rectus muscle was observed for the first time; no facial palsy was present. On the second postoperative day, the patient was generally improved; the left abducens palsy remained unchanged. On the third postoperative day, the child's temperature fell to below 100 degrees, where it remained for the rest of the hospital stay; the patient at this time appeared well, alert and bright and remained so until the time of discharge. The left abducens palsy improved steadily, so that at discharge it was almost absent. The operative wound healed rapidly, granulating in most satisfactorily. The discharge from the wound was only moderate and presented no unusual features; the discharge from the ear ceased by the time of the patient's discharge from the hospital.

At the time of completion of this paper (July 8, 1938), the patient was perfectly well, the wound healed and the left abducens palsy entirely absent.

LABORATORY DATA

Urine negative.

Blood count taken after operation showed a moderate leucocytosis with a slight shift to the left and a high toxic index; no eosinophilia.

Pathology report on the tissue removed at operation: "Fragments of necrotic bone showing evidences of acute inflammation."

Bacteriology report: The smear taken from the mastoid bone at operation revealed "very occasional degenerated pus cells and moderate Gram positive pleomorphic bacilli." Cultures taken at the same time showed after 24 hours "Gram positive forms, probably a member of the group of higher bacteria."

Cultural studies were then carried out to the conclusion that the organism was a member of the genus actinomyces. This opinion was concurred with by Dr. C. W. Emmons, senior-mycologist of the United States Public Health Service.

Bacteriological Studies: During the time the organism was studied, the cultures were at all times pure and uncontaminated.

(a) Morphological Characteristics: The most striking feature, other than the mycelia, was the pleomorphism which the organism demonstrated. In actively growing cultures 24 to 48 hours old, the morphology was quite uniform. They were short and long thick Gram positive bacilli with a few spiral and crescentic forms. In older cultures one found simultaneously coccoid, bacillary and diphtheroid forms. The ends of the latter two frequently presented round or pear-shaped swellings. Branching appeared in five to six days, but was never striking, apparently because of the ease with which the mycelia fragmented.

(b) Cultural Characteristics: The organism was quite remarkable for the facility with which it could be subcultured for the several months during which it was studied, and for the ease with which it grew in aerobic and anaerobic media. The organism grew very well on DH agar, Sabouroud's medium, starch agar, honey agar, gelatine, dextrose and blood agar; growth in broth was very poor. It did not liquefy gelatine.

The organism was not acid-fast.

(c) Animal Experiments: Thirty mice were injected intraperitoneally with one cubic centimeter each of a suspension of the organism with no effect. An adult male rabbit was given five cubic centimeters of the suspension intravenously, intramuscularly and intraperitoneally; after two months the rabbit was killed, but examination failed to reveal any actinomycotic lesions.

REVIEW OF THE LITERATURE

A review of the literature attests to the rarity of involvement of the mastoid bone. As we will see, the majority of pertinent case reports describe actinomyces mastoiditis as but an incidental feature in a more extensive actinomycotic infection, rather than as a primary lesion.

The earliest case of actinomycotic infection of the mastoid bone was reported by Ginsberg in 1890.[8] His case was one of a 30-year-old male, who presented a history of pain in the left ear which was

followed by a spontaneous rupture of the ear drum with discharge of pus; shortly afterward marked cervico-facial swelling appeared. This was incised and drained with almost complete relief. Five months later, the swelling reappeared associated with a left facial palsy and terrific headache. The patient failed rapidly and died in a few days. Postmortem examination disclosed the actinomyces in the left ear, left mastoid and temporal bones and the tonsils.

Two years later Majocchi[9] described a case in a middle-aged male, with mycosis fungoides, who developed a right otitis media followed by headache and proptosis of the right eye, terminating rapidly in death. Necropsy revealed actinomycotic involvement of the mastoid bone with basilar meningitis and thrombosis of the transverse sinus.

Dor,[10] in 1893, reported a case in which, following the extraction of a tooth, there appeared a left cervical abscess which was succeeded by a left otitis media discharging pus in which the actinomyces was found; shortly after this mastoid tenderness appeared with a soft painful tumor just below the mastoid process. The mass opened and discharged pus containing the actinomyces. While mastoiditis was present clinically, we cannot consider this case with certainty as one of actinomyces mastoiditis.

Reinhard[11] reported a case of purulent otitis media with mastoiditis, operated on and followed by a Bezold's abscess; examination of the pus revealed the actinomyces. The details of the case were not given. Ten Siethoff[12] in discussing this case, mentioned a similar one that he had treated with iodides with complete recovery in four weeks.

DeQuervain's case[13] was interesting; his patient presented an intermittent right otorrhea complicated by an acute right mastoiditis which was temporarily relieved by a mastoidectomy. This was shortly followed by a left otitis media and mastoiditis due to the actinomyces; this was soon after confirmed by necropsy which, in addition, disclosed actinomycotic involvement of the base of the brain and the meninges. Whether or not the original infection in the right ear was due to the actinomyces was not known and remained a point for speculation.

The cases reported by Littledale[14] and Urbanschitch[15] on actinomycotic otitis media with tender masses over the mastoid process cannot be accepted as unequivocal instances of mastoid involvement.

Beck's case[16] was the first reported in this country, although the patient was seen in Zaufal's clinic in Europe. The patient was a

middle-aged farmer with a mass behind the left ear and over the mastoid process of some six months' duration; the mass spontaneously ruptured discharging characteristic actinomycotic granules. Death occurred suddenly after several months of observation; necropsy disclosed actinomycotic infection of the mastoid and temporal bones with rupture of the right vertebral artery.

A case of "primary" acute actinomycosis of the mastoid was recorded by Bruzzone;[17] details of the case were not given other than that the patient, a 48-year-old male, fell and suffered many skin abrasions, as a result of which a fatal septicemia followed. During the course of the illness, the clinical picture of an acute mastoiditis appeared; the actinomyces was isolated from the involved bone.

Drury[18] in a paper entitled, "Mastoiditis Actinomycosis," described in detail a case of a 35-year-old female in whom, following the extraction of several infected teeth in the left upper jaw, there appeared tenderness and swelling above the left ear. In the following eight months several abscesses about the ear were operatively drained; then a mastoidectomy was done with the removal of necrotic bone. A short time following operation the actinomyces was isolated from the discharge for the first time. The patient died after a long illness.

DISCUSSION

A review of the case history presents the problem as to the mode of infection; the pathogenesis of acute actinomyces mastoiditis, and actinomycotic infections is therefore pertinent.

Bostroem[2] described a member of the genus actinomyces which he had isolated from actinomycotic lesions in cattle and from grasses upon which the cattle grazed. Because of these findings and because of the fact that the common lesion in cattle was of the cervico-facial type, Bostroem concluded that the infection in cattle and man was due to the ingestion of grains and other vegetable matter infected by the actinomyces which, as a result of the trauma produced during mastication, invaded the buccal tissues. This opinion, which for convenience we may term the "exogenous" theory, was held for many years and is still being taught by many.

The "exogenous" theory has been opposed, notably by Lord, Naeslund and Emmons, who contend that the actinomyces is an inhabitant of the normal alimentary canal, particularly the mouth, and that this organism may under unfavorable conditions invade the tissues of the host and produce lesions. This concept, the "endogenous" theory, is supported, we believe, by careful and conclusive studies.

Thus, Lord[19] in 1910 isolated from tonsillar crypts and carious teeth of healthy individuals an organism with the morphology and staining characteristics of the actinomyces; this when injected intraperitoneally into guinea pigs gave rise in 60 per cent of the animals to omental tumors with typical actinomycotic lesions from which actinomycotic granules were recovered. Naeslund[20] was successful in isolating from the mouths of normal individuals an organism similar to that described by Israel which was capable of producing characteristic lesions in experimental animals. Emmons,[21] in a careful study of 100 pairs of tonsils removed at routine tonsillectomies, observed the actinomyces upon direct examination in 47 per cent of the specimens, while in 10 per cent the actinomyces bovis was obtained in pure culture.

The clinical evidence supporting these two concepts favor strongly the "endogenous" theory. The concept of "exogenous" infections was believed confirmed by the occasional finding of straw, grain, etc., in the lesion; this foreign body, we believe today, was the traumatic agent facilitating the invasion of the tissues by the actinomyces inhabiting the mouth rather than the means of introduction of the invader.

Furthermore, we find clinical reports clearly demonstrating the "endogenous" theory; thus cases have been reported of actinomycotic infection following instrumentation of the buccal cavity, as in dental extraction.[22] In addition, a number of cases following human bites have been recorded.[23]

We know today that the organism described by Bostroem was an aerobe, a saprophyte widely distributed in nature and seldom pathogenic for man; on the other hand, the organism usually responsible for human actinomycosis is that described by Israel, a microaerophilic form usually found in the alimentary canal.

The status of the problem of pathogenesis may be summarized as follows: actinomycosis in man is caused by the actinomyces, usually of the bovis species, a microaerophilic organism existing as a harmless saprophyte in the alimentary canal and invading the body tissues only under unfavorable conditions, such as may follow local trauma, diminution of local tissue resistance and increase in the pathogenicity of the organism.

Thus, on the basis of the "endogenous" theory, we may clarify the case reported by postulating the occurrence of a primary actinomycotic infection in the mouth or nasopharynx with secondary

extension to the middle ear by way of the eustachian tube with a subsequent progression to the mastoid bone.

When one considers the frequency with which the actinomyces can be isolated from the normal mouth, it is surprising that it has been reported so rarely in otolaryngological infections. It is very likely that the incidence of such infections is higher than is evidenced by the reports in the literature because of our failure to be alert to their occurrence and because of the difficulties usually encountered in the laboratory in culturing and studying the organism, particularly when it simulates the common pathogenic bacteria.

Appreciation of the occurrence of such actinomycotic infections should lead to greater care and effort in the bacteriological studies of mastoiditis.

SUMMARY

1. A case of acute actinomyces mastoiditis treated by simple mastoidectomy with complete recovery is reported. As far as can be ascertained, this is the first case of its kind reported.

2. A review of the pertinent literature is presented.

3. The pathogenesis of actinomycosis in man and in the reported case is discussed.

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XX

MALIGNANT TUMORS OF THE NASOPHARYNX AND PARANASAL SINUSES*

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The treatment of malignancies of the nasopharynx and paranasal sinuses comprises but a small part of the otolaryngologist's practice, yet these cases often prove distressing, chiefly because of delay in diagnosis, problems in treatment and results.

Confusion has been the keynote in the nomenclature until recent years, when New, Broders and Childrey[1] declared that nasopharyngeal malignant tumors could be largely classed as of two types, squamous cell epitheliomata of high grade malignancy and lymphosarcoma. They found epithelioma occurring seven times as frequently as sarcoma. The tumors previously designated as anaplastic, epidermoid and transitional cell carcinoma, endothelioma and lympho-epithelioma were thus grouped together as squamous cell epitheliomata.

Recently Salinger and Pearlman[2] submitted slides from 24 malignant tumors of the epipharynx to three pathologists. The diagnoses of the latter showed remarkable agreement. These data and a study of the literature led the authors to conclude that anaplastic undifferentiated and epidermoid carcinoma could be condensed into one, the transitional cell type including so-called lympho-epithelioma; that aside from Broders' classification most of the terminology has been in error.

Tumors of the sinuses are more varied in type, but a condensation of terminology might well also be applied here, since the terms epidermoid, anaplastic and transitional cell carcinoma are frequently encountered. However, true squamous cell epithelioma with pearl formation is by no means rare. Sarcoma of the usual types is often seen and basal cell and adenocarcinoma occur occasionally. Moreover, the sinuses may be the seat of more unusual types of malig-

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nancy, such as osteosarcoma,[2] plasmocytoma,[4] rhabdomyoma,[5] hypernephroma,[6] teratoma,[7] or the mixed type of carcinoma.[8]

The maxillary and ethmoid sinuses are the most frequent seats of malignant tumors, while primary neoplasms of the sphenoid and particularly the frontal sinuses are rare.

Nasopharyngeal malignancy may occur at any age, although most commonly between the ages of 40 and 60 years. The focus of the tumor is often small, one of its chief characteristics being to spread widely and deeply beneath the mucous membrane so that severe intracranial symptoms occur early, while ulceration, hemorrhage and nasal obstruction are late manifestations. Exception may be taken to this in the case of lymphosarcoma, whose tendency to large bulky growth produces nasal obstruction as the most common early symptom.

The tumor is most frequently located on the lateral pharyngeal wall, producing obstruction of the eustachian tube, with pain in the ear, tinnitus, impairment of hearing and aural discharge. These signs, together with pain and parathesias in the distribution of the fifth nerve and cervical adenopathy, produce a significant and early triad of nasopharyngeal malignancy, the occurrence of any one of which should direct attention to the pharyngeal wall. New,[9] in a series of 246 cases found cervical adenopathy to be the first symptom in 46 per cent, cranial nerve palsies in 31 per cent. Extension of the tumor may be into any of the sinuses, occasionally into the anterior cerebral fossa, causing involvement of the second, third, fourth, fifth and sixth cranial nerves. The neurological symptoms have been well described by Waltman,[10] New,[11] Burger,[12] and others.[13] New emphasized the importance of nasopharyngeal examination in patients having unexplained cranial nerve palsies.

The tumor may extend back into the tissues of the pharynx, compressing the ninth, tenth, eleventh and twelfth cranial nerves and giving rise to Jackson's syndrome, complete or partial. Or extension upward may involve the hypophysis and optic nerves, suggesting brain tumor. Involvement of more than the fifth cranial nerve means intracranial extension and almost hopeless prognosis.

Schlivek[14] discussed the symptoms referable to the eye and listed the ocular signs as follows: Involvement of the fifth, sixth and seventh nerves, Horner's syndrome, ophthalmoplegia interna and externa, exophthalmos, ptosis, papilledema, papillitis, and optic atrophy. He suggested an ocular syndrome of nasopharyngeal malig-

nancy, namely, involvement of the fifth, sympathetic and sixth nerves, either individually or together, with symptoms referable to the ear.

Any of the above signs or symptoms may occur before the tumor, even though suspected, is found. A bulging area, especially of the lateral wall of the nasopharynx, though it be smooth and regular, demands a biopsy. Occasionally malignancy may be discovered at tonsillectomy, particularly in adults, by routine palpation of the nasopharynx. Thus, we inadvertently discovered a squamous cell epithelioma in a man of 50 years which was as yet completely asymptomatic. This tumor disappeared completely under intensive deep x-ray therapy. Failure to consider the nasopharynx in cases of conduction deafness, cranial nerve palsies, cervical adenopathy and ocular signs may result in diagnostic error.

Malignant tumors of the nasal sinuses show in general the same age and sex qualifications as noted for nasopharyngeal malignancies. Regional metastases are less common than in nasopharyngeal tumors. Peyton[15] found two with palpable regional nodes in a review of ten cases of squamous cell carcinoma of the maxillary sinus. Here again the great tendency is for local extension of the lesion, so that frequently more than one sinus is involved and determination of the original focus is not always possible. Hemorrhage is the most frequent early symptom in tumors of ethmoid origin and this is soon followed by nasal obstruction. Pain about the upper teeth and in the face direct one's attention to tumors of the antrum. Teeth are often wantonly extracted during this stage of the disease. Bulging of the cheek, bulging of the hard palate or proptosis of the eye should at once suggest sinus neoplasm in the differential diagnosis. That a previously satisfactory dental plate no longer fits is a frequent early complaint in patients with malignant invasion of the maxillary antrum. Blackwell[16] pointed out that bloody mucous discharge with no accompanying high blood pressure should institute a search for nasal malignancy. Transillumination and the roentgenogram, which may show bony erosion may be of great aid in the early diagnosis of these tumors.

Barnes[8] and Hill[17] noted the frequency with which polyps had previously been removed from the sinuses and recurred manifestly as a tumor. These authors and Robinson[18] made a plea for early exploratory operation in all suspicious cases and microscopic examination of all but the most innocent material removed in operations upon the sinuses.

A history of the pre-existing chronic infection is so often obtained that Barnes,[8] Wright[19] and Sharp[20] emphasized this as a factor in the etiology of the condition.

The presence of borrelia vincenti in a fistula or ulcerated extension into the mouth of an antral tumor should not obscure the diagnosis, since it is possible to find this organism in almost any ulcerated lesion about the mouth such as in agranulocytic angina, leukemia, lymphosarcoma or carcinoma.

Although Beck and Guttman[21] and New, Broders and Childrey[1] have stated that the radiosensitivity of malignant tumors of the upper respiratory tract is determined by the histological structure of the tumors, Martin[22] and others[23, 24, 25, 26] have noted that the response to radiation of new growth in this region does not correspond to the theoretical radiosensitivity based on the histology. In Martin's successfully irradiated group of nasopharyngeal tumors, 55 per cent showed an adult cell structure and 45 per cent were of the anaplastic variety.

There is only one treatment for malignancy of the nasopharynx, and that is irradiation. Usually both radium and roentgen therapy are employed. Complete cures are often prevented by the presence of metastases or intracranial extension, but the general improvement which so often follows irradiation makes it worthwhile anyhow. Martin gave an excellent summary of irradiation therapy, and Cutler[26] showed some very promising results with the radium pack. New, Broders and Childrey[1] obtained 18.28 per cent of five year cures in 32 patients, Coutard[27] reported 10 per cent in 89 cases, and Martin[23] 35 per cent of 21 patients cured for periods of one and a half to three and a half years.

The modern treatment of malignant tumors of the sinuses shows somewhat better results, as noted in Table I. There, irradiation alone or irradiation and diathermy combined gave the best results. Highly radio-sensitive tumors such as lymphosarcomata were treated only by irradiation, although an operative approach was added in many cases for radium implantation and to facilitate drainage. In the majority of reported cases the tumors were destroyed by diathermy, then treated by irradiation. New is a leader in this field. In a recent article[32] covering a study of 295 cases of malignancy of the upper jaw and antrum operated upon prior to 1929, he reported 53.8 per cent five-year cures. His procedure for treatment of antral malignancy is as follows: Nitrous oxide intratracheal anesthesia, alveolar or palatal approach and biopsy and frozen sections for diag-

TABLE 1

SUMMARY OF RESULTS OBTAINED IN MODERN TREATMENT OF MALIGNANT TUMORS OF THE SINUSES

Date	Author	Type of Therapy	Locality	No. of Patients	Percent Cured	Duration of Cure
1932	Peyton	Irradiation Irradiation and diathermy	Antrum	11	10	5 years
1934	Watson-Williams (28)	Radium	All sinuses	10	90	3½ years
1934	Öhngren (29)	Diathermy and Irradiation	Antrum	52	38.5	5 years
1935	New and Cabot (30)	Diathermy and Irradiation	Antrum	91	40	5 years
1935	Harmer (31)	Diathermy and Irradiation	Antrum	49	12.2	5 years
1935	Robinson	Radium plus surgery	All sinuses	60	20	5 years

nosis. Then tumors of low grade malignancy are destroyed entirely by diathermy followed by radium or roentgen-ray therapy. Monthly postoperative observation is emphasized.

When the growth extends into the ethmoids or beyond, Barnes and Hill prefer the more radical Moure incision followed by irradiation. However, some of Robinson's[18] good results were obtained with radium alone and many will prefer this method.

We have no large series of cases from which to draw conclusions. Three case reports are selected from our group which are typical examples of nasopharyngeal and nasal sinus malignancy, considering type, location and symptomatology.

REPORT OF CASES

CASE 1. S. T., a white man, aged 47 years, was first seen on March 13, 1936, with humming in the right ear of one year's duration, numbness of the right side of the face for six months, some pain in right eye and swelling of the upper right eyelid for two weeks. The humming, which was accompanied by

some deafness, had started as a crawling sensation in the right ear. The numbness of the cheek had been accompanied by a drawing sensation, soon followed by a dull aching pain which seemed to be in the bone. He also complained of numbness of the tip of the tongue on the right side and occasional diplopia. Intermittent discharge of watery fluid as if something "breaking loose" was noted. Examination showed ptosis of right eye with slight proptosis and interference with movements to the left. The patient maintained rotation of head to the left to avoid diplopia. The pupil was miotic and reacted promptly to light and accommodation. The vision was normal and the fundi were negative. Hearing was markedly decreased in the left ear (spoken voice at three feet). Both bone and air conduction were diminished. Examination of the nose, nasopharynx and throat was negative. Transillumination and x-ray of sinuses were negative. Sensation of the right side of the face was reduced. An area of hyperesthesia surrounded the shoulder girdle. There were no cervical nodes palpable at this time or during the entire course of his illness. Rhomberg's sign was present, the biceps reflex was decreased on the right and the patellar reflex was exaggerated bilaterally. With this complex picture and in spite of a negative spinal fluid, syphilis of the central nervous system was suspected and the patient was placed on the therapeutic test. Diagnostic operative opening of the posterior ethmoid, antrum and sphenoid was negative. Nothing could be felt by deep palpation of the orbit and the eye could be replaced backward into position easily. There was no response to treatment, and as paresis of all extra-ocular muscles increased, especially those muscles supplied by the external branch of the third nerve, a deep orbital tumor was diagnosed. This diagnosis could not alone explain the whole symptomatic picture plus a noticeable change of personality and mentality. Repeated nasopharyngeal and sinus examinations were made, and on July 17, 1936, a small tumor was palpated in the region of Rosenmüller's fossa, right side. The swelling was smooth, sessile and hard. Biopsy was done with difficulty because of the size and inaccessibility of the growth. Our pathologist reported epidermoid carcinoma, grade 2. The patient was admitted to the hospital and three radon seeds of 3.04 m.c. each were implanted, two in the retro-bulbar area and one in the growth in the nasopharynx. These were left in position ten days. The patient died January 2, 1937, and at autopsy the tumor was found originating in the right nasopharynx and involving the apex of the orbit, the optic foramen, the petrous portion of the temporal bone and the anterior fossa of the skull, with softening of the frontal lobe of the brain.

This case fulfills the criteria for Schlivek's ocular syndrome and well illustrates the invasive, burrowing extension of the tumor into the tissues and bone while the primary lesion in the nasopharynx remains small and hard to find. The diagnosis should have been made earlier than it was and a larger dosage of radium or radium plus deep x-ray could have been given when there still was a possibility, even if not a probability, of cure.

The following case illustrates the marked symptomatic improvement and the profound effect produced upon these tumors, even when hopelessly extensive, by radiation.

CASE 2. A. S., a white man, aged 61, was admitted to the hospital on February 11, 1938, because of nose-bleeds and a mass filling his right nostril. The first sign of nasal pathology was evidenced in August, 1937, by a severe attack of epistaxis from the right nostril which was treated by the family physician. The patient complained of nothing further until November, 1937, when he began

to have pain in the right eye and right frontal headache. In January, 1938, the patient noticed swelling of the right upper lid, diplopia and difficulty in moving the right eye. The right nostril was obstructed. The patient became lethargic, weak and depressed about one month before admission. His wife noted a definite personality change. He was referred to the hospital, his physician stating that biopsy upon the growth in the nose showed carcinoma. On admission he was in a stuporous condition. There was edematous swelling of both lids of the right eye and chemosis of the conjunctiva with incomplete external ophthalmoplegia. The right nostril was filled with a large friable, easily bleeding tissue which extended from the lateral nasal wall. A roentgenogram revealed clouding of the right antrum and both frontal sinuses. The pathologist reported grade 3 carcinoma on biopsy.

On February 15, 1938, roentgen therapy was instituted. Six thousand roentgen units were given to the nose and sinuses over a period of twenty days. The patient was discharged on April 7th, feeling and looking remarkably improved. He was much stronger, alert and cheerful. The tumor mass in the nose had melted away and the sinuses transilluminated clearly. He died at his home six months later of brain complications—no doubt malignant.

The following case illustrates the early rapid growth and prompt recurrence of a fibrosarcoma of the posterior ethmoids in a young boy.

Case 3. D. J., a white boy, 5½ years of age, came to the Guthrie Clinic on February 17, 1938, because of nasal obstruction and mucous discharge on the left side, of one month's duration. Examination showed a tumor mass high in the vault of the nose and in the region of the posterior ethmoids. It was firm, immovable and under pressure, as though jammed in tightly against the posterior septum. There was no cervical adenopathy. Biopsy of the tumor showed fibrosarcoma, grade 2. The tumor was removed through the pharynx and left a cup-like depression in the bony septum. The size of the tumor was 2x3 cm. approximately. Ten days later deep x-ray treatments were started. He was given 6,000 roentgen units to the posterior nasal area through three portals over a period of twenty days. He was seen monthly and the involved area was clean and normal in appearance until November 15, 1938, a recurrence in the form of a small nodule was noted. He was again placed upon x-ray treatment, 6,000 roentgen units repeated with the same technique. On December 20, 1938, no evidence of growth could be seen.

Owing to the age of the patient and because of the early recurrence of the growth the outlook is poor.

SUMMARY AND CONCLUSIONS

1. Three case reports were selected from a group because of some special illustrative point.

2. It appears from a review of the large experience of others that more and more importance is placed on irradiation, regardless of classification of the tumor, which is of secondary importance and sometimes misleading in regard to applied therapy.

3. Irradiation is becoming more intelligently and effectively used.

4. The more highly malignant these tumors of the nasopharynx and the nasal sinuses are, the more reliance must be placed on irradiation.

5. Early diagnosis and intensive treatment are all-important in the hope of obtaining the highest percentage of cures.

6. A simple standard terminology will help to clear the confussion which still exists in the classification of these tumors.

The author is indebted to Doctors Charles H. Scheifley and David P. Cordray for their help in collecting the data for this paper.

ROBERT PACKER HOSPITAL.

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XXI

SOME EXPERIENCES WITH A VITAL STAIN ON OTOLARYNGOLOGICAL TISSUES*

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The correction of pathology in the ears, nose and throat by medicinal agents is our chief concern. Usually very little of this pathology is on the surface or in the first layer of cells. Therefore, our medicines must penetrate tissue to reach the intended site of action.

If we knew all the laws that govern the passage of substances through tissues, a large fraction of the mysteries of life would be solved. Why are certain substances and not others absorbed from the digestive tract? Why do special membranes excrete certain compounds and not others? Why do the cells of certain glands secrete the same compounds throughout life? Why do nerve cells admit certain compounds for nutriment and muscle cells admit others? Such questions as these might be continued almost indefinitely. On each of such questions a multitude of scientists have worked for many years. The result of their labors has been the discovery of a relatively few chemical formulæ and reactions and a few laws of ionization, osmosis, imhibition, etc. The workers are to be commended, but still we know relatively little of the reasons for the transmission of substances through tissues.

Many ingenious methods have been employed to measure this permeation of the tissues. The qualitative and quantitative measurements on the two sides of a tissue or tissues over a period of time have assisted much in the problems of the digestive tract and of the kidney. The microscopic examination of specially stained sections of intestine has assisted in the problem of absorption of fats. The appearance of characteristic physiological changes after the administration of certain substances by various routes has indicated the rate and degree of absorption. Epinephrin, sodium nitrate or digitalis

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†Deceased, December 21, 1938.

passing through a multitude of different cell layers, produces characteristic changes in blood pressure, heart rate and respiration.

To measure the depth of a substance's penetration into tissue, investigators have used colored chemicals. Much has been learned about the absorption of fats from the intestine by staining sections of the intestine with osmic acid following a period of absorption. The extent of penetration of the skin by mercury has been studied by precipitating the mercury with a sulphide and observing the result under the microscope. Weed used potassium ferrocyanide and iron ammonium citrate to inject tissues, which were later acidified, resulting in the precipitation of Prussian blue. Recently in our own field Hodjakoff used trypan blue in the middle ear. He found that it extended deeply into the structure toward the inner ear.

There are certain disadvantages in all of the methods of determining the depth of absorption that depend on the visualization of the substance in the tissues. First, the color of the substance may not be dense enough in minute quantities to be detected. Second, it may not be possible to make the substance insoluble at any desired moment and thus prevent postmortem travel of the compound in dead tissues.

After a study of various substances, mercurochrome was tried as a vital stain. The term "vital stain" is used here to mean a non-injurious substance which stains living tissues and whose spread can be stopped at any desired moment. It is difficult to classify mercurochrome as either an intravitam or a supravitam stain. Vital stains are usually injected intravenously, resulting in the deposit of the stain in certain types of tissue throughout the body. At present, we are interested only in the spread of a substance in living tissues. Mercurochrome is soluble, non-necrotic and non-coagulating. Therefore, it should be absorbed quite easily, with the tissues remaining in their natural state. Dilute mineral acids will precipitate mercurochrome. The precipitate is a brilliant yellowish-red substance which is soluble in the common organic solvents. Therefore, it is possible to apply mercurochrome to a tissue for a period of time, then perfuse the animal with normal salt solution containing two to three per cent hydrochloric acid, precipitating all of the absorbed mercurochrome. If the tissue is bone, decalcification may be carried out without harming or removing the stain. The common methods of dehydration cannot be employed because of the solubility of the acid-mercurochrome in alcohol and other dehydrating agents. We

have used frozen sections which were examined either wet or air-dried and mounted in the usual way.

For some time we have been interested in the passage of substances from the middle ear to the inner one. It has been demonstrated that many compounds do pass through. It was thought that by using mercurochrome as a vital stain, that the chief route of passage might be discovered.

Dogs were anesthetized, and an eight per cent solution of mercurochrome was placed in the middle ear. At the end of different periods, twenty minutes, forty minutes, one hour and two hours, the animals were perfused with three per cent hydrochloric acid in normal salt until the return flow was quite clear. Then the perfusion was continued with three per cent hydrochloric acid in five per cent formalin solution. In some cases this perfusion was continued intermittently for one to three days. The temporal bones were then taken out and decalcified in the usual way.

We have prepared a couple of colored microphotographs of sections of a temporal bone which have been penetrated by mercurochrome. These sections were typical of those obtained from eleven ears. It was clearly seen that the mercurochrome in large amounts passed through from the middle to the inner ear. The tissues named in the decreasing order of depth of stain were: (1) bony wall of middle ear; (2) fenestra rotunda; (3) nerve cells of the spiral ganglion, spiral lamina, and eighth nerve; (4) stria vascularis; (5) articulation of the stapes, and (6) meshes of the aqueduct of the cochlea. These results seem to indicate that the fenestra rotunda is not the chief avenue of passage for mercurochrome into the inner ear, but that the thinner bony walls carry a great deal of it.

Several dogs were anesthetized and fixed so that the nose was extended horizontally with the head turned about 45 degrees to the right. Several cubic centimeters of eight per cent mercurochrome were put into the right nostril. The mercurochrome was allowed to penetrate for one or two hours. At the end of the period, the usual perfusion and fixation were completed.

A colored microphotograph of a section of a turbinate exposed to the mercurochrome for one hour shows that the stain passed entirely through it.

Another specimen, the exposed frontal bones of an animal treated as has been described, showed that the mercurochrome has

penetrated the outer plate of the frontal sinus sufficiently to show grossly.

It was shown that the mercurochrome had passed into the internal walls of the frontal sinus to show the stain through the dura. The cortex of the brain in this area was distinctly colored with mercurochrome. The penetration of mercurochrome through the walls of the frontal sinus was not grossly visible in all cases; therefore, final conclusions cannot be drawn.

In conclusion, we wish to emphasize several things: First, mercurochrome can be utilized as a vital stain to add to our knowledge concerning the penetration of otolaryngological tissues by therapeutic substances. Second, our work in this field is only introductory and final conclusions are not yet permissible.

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XXII

ACUTE LARYNGO-TRACHEO-BRONCHITIS: REPORT OF THREE RECOVERED CASES

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The subject of acute laryngo-tracheo-bronchitis is of paramount interest to oto-rhino-laryngologists, pediatricians and general medical practitioners. The fact that the most expert efforts often fail to change the fatal trend of this malady has served in the past to make some of us despair of attempting to do anything. It is, therefore, extremely important that any measure that seems promising should be given a thorough trial, even though its mode of action is not entirely clear.

The present communication deals with three recovered cases, two of staphylococcus albus origin treated with staphylococcus bacteriophage, and one due to streptococcus hemolyticus.

PRESENT STATUS OF OUR KNOWLEDGE OF THIS CLINICAL ENTITY

Etiology: Acute laryngo-tracheo-bronchitis is an inflammation of the mucous membrane of the larynx, trachea and bronchi, usually preceded by symptoms of a head cold. It may, however, be primary in the larynx or subglottic region. Occasionally it follows the aspiration of a foreign body into the trachea or bronchi. It is a disease strikingly prevalent in infants and young children, although recently one adult case of hemolytic streptococcus origin was seen at the Massachusetts Eye and Ear Infirmary, and another was reported by Gittins.[1] Direct examination of smears taken from the larynx and trachea usually reveal a mixed infection of staphylococci, streptococci, and pneumococci, but invariably, cultures will produce growths of pure streptococci or staphylococci.

Gross Pathology: This depends on the existing causative organisms. In all types of infection, especially during the early stages, there is an inflammatory reaction of the larynx, involving chiefly the epiglottis, false cords and arytenoid regions. In the streptococcus type it is associated with marked fiery redness and swelling of the subglottic and tracheal regions, together with a pronounced velvety appearance of the tracheal mucosa. The true cords, however, may

show comparatively little reaction. As the disease progresses, necrotic gangrenous pseudo-membranes may be seen extending down to the tracheal bifurcation and into the main bronchi. The subglottic swelling causes mild dyspnea early in the disease. Later the obstructive gangrenous pseudo-membrane is responsible for more pronounced dyspnea, often extreme and leading to a surgical emergency.

If, however, the causative agent is staphylococcus, the inflammatory reaction of the tracheal and pharyngeal mucosa is practically always accompanied by thick, dirty-grey, tenacious mucous plugs which tend to dry and crust, and rapidly cause marked progressive obstructive symptoms. Frequently some of these crusts can be seen early in the infection, covering the vocal cords and obstructing the glottis. Removal of the tracheal and bronchial crusts does not reveal any necrosis of the tracheal mucosa. This, however, is frequent in the late streptococcic type.

Symptoms: The disease may be ushered in by an infection of the upper respiratory tract with its associated symptoms and then soon followed by fever, hoarseness, dry croupy cough and rapidly progressive dyspnea. In the streptococcic infections the elevation of temperature is usually higher than in the staphylococcic type.

As the dyspnea rapidly increases in severity, marked retraction of the suprasternal fossa, the supraclavicular spaces, the epigastrium and marked depression of the intercostal spaces appear. This is accompanied by marked pallor and cyanosis. At this stage auscultatory examination of the chest may reveal failure of air to enter the lungs, due to obstruction of the bronchi by dry, crusted mucous plugs. The final picture is, as ably described by Richards,[2] a ghastly spectacle of death by asphyxiation and suffocation.

Such is the usual course of the disease when unrelieved by treatment, death often occurring thirty-six to forty-eight hours from the onset of the laryngeal symptoms.

REPORT OF CASES

Case 1. M. M., a boy, age 20 months, was admitted to the private ward of the Massachusetts Eye and Ear Infirmary on the afternoon of November 22, 1936. The parents stated that two nights previously the child developed slight hoarseness without any apparent previous symptoms of a head cold. The morning before admission he awoke with a harsh, dry, barking cough and a temperature of 102 degrees. The local doctor was called, who advised rest in bed and prescribed a cough mixture. The next morning, i.e, on the morning of the day of admission to the hospital, the patient awoke feeling very much better, seemed quite cheerful and playful, had a normal temperature, but refused to eat any breakfast.

At 12:30 that afternoon he suddenly developed a very hoarse cry, together with marked difficulty in breathing, and then threw his head back and became quite

motionless and cyanotic. The local doctor was again called, and advised admission to the hospital for immediate tracheotomy. On admission the child presented extreme inspiratory and expiratory dyspnea with marked supraclavicular and intercostal retraction, and marked cyanosis. X-ray of the chest revealed a narrowing of the trachea and suggested the presence of a mediastinal abscess.

Direct laryngoscopy was attempted, but the entire larynx was covered with a thick, crusted, tenacious secretion and the patient ceased breathing. A Mosher straight life-saving tube was inserted between the cords, and without using any anesthesia, an emergency low tracheotomy was performed and the tracheal opening enlarged with a small ring punch.

On making the incision to the trachea, a small amount of pus exuded from the pretracheal region just above the sternum. Marked relief of the dyspnea immediately followed the tracheotomy and the normal color of the patient was soon restored. No bleeding being encountered, a smear and culture from the tracheal mucosa were taken, and a No. 4 short tracheotomy tube was inserted. The patient was returned to his room and placed in a compound tincture of benzoin croup tent. The direct smear showed a gram positive diplococcus with a few short chain streptococci and many staphylococci, but a culture proved to be a pure gram positive staphylococcus. The following day the patient seemed quite comfortable, breathed freely and drank fluids well, but refused all foods.

The next morning, November 24, the child awoke with a dry cough and slight dyspnea, and by noontime had developed fairly marked difficult breathing with slight cyanosis. Auscultatory examination of the chest by the attending pediatrician was negative and immediate oxygen tent with ample morphia for complete rest were advised. Fluoroscopy of the chest revealed air freely entering and leaving the lungs, and x-ray showed slight thickening of the right base and a clear mediastinum.

About four hours later, in spite of the oxygen tent and morphia, the dypnea had so rapidly increased that the respirations became labored, rapid and shallow with very marked supraclavicular and intercostal retraction and extreme cyanosis. The child was actually moribund. The oxygen tent was of no value, for auscultation of the chest revealed no air entering or leaving the right lung and also partial obstruction of the left lung. The patient was taken to the operating room and a bronchoscope was inserted through the tracheotomy opening. Completely occluding the right main bronchus was a hard, dry, adherent, dirty brown crusted mass. Several attempts to soften and to dislodge it, using solutions such as saline, adrenalin, and sodium perborate failed. Finally the mass was grasped with an alligator-tooth forceps and with some force was removed together with the bronchoscope, but there was no improvement in breathing. The bronchoscope was again passed and after some difficulty, the bronchi, leading to the right upper and middle lobes, were also cleaned of similar hard, dry, crusted masses. Immediately following this, the breathing became less labored and the patient was much more tranquil. A few minutes later the bronchoscope was again inserted through the tracheal opening to the left main bronchus, which was found partially occluded with a similar crusted mass. This was easily removed.

At this point the writer discussed with Dr. Heine, who was serving as house officer at the Massachusetts Eye and Ear Infirmary, the results he obtained in his treatment of staphylococcus osteomyelitis of the frontal bone with staphylococcus bacteriophage. The results of its use encouraged the writer to try it in this case,

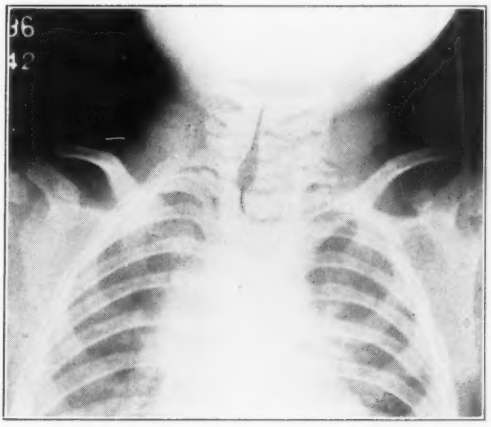

Fig. 1. Antero-posterior view, showing the trachea displaced to the right and compressed by an abscess in the lower left neck.

which was also due to staphylococcus organisms. About 8 ccs. of staphylococcus bateriophage was instilled into each lung and soon removed by suction. The patient, now breathing normally, was transferred to a small private room, and placed in a benzoin steam croup tent.

At frequent intervals, about 5 ccs. of staphylococcus bacteriophage was instilled, through the tracheotomy tube, into the right lung. Each instillation of the fluid caused a fit of coughing with expulsion of a large amount of thick, viscid, purulent discharge, which was removed by suction through the tracheotomy tube. The same procedure was carried out with the left lung. From then on to the time of the removal of the tube there was a gradual reduction of the cough and sputum, and a marked general improvement.

Ten days later the tracheotomy tube was removed, and on December 13, three weeks after admission, the patient was discharged from the hospital, the tracheotomy wound healed. The lungs were clear and the speaking voice normal but still with slight hoarseness on crying.

Comment: This case presents the typical course of staphylococcus laryngo-tracheo-bronchitis developing without any apparent antecedent upper respiratory infection. The rapidity of the change in the patient's clinical state from relative wellbeing to extreme respiratory difficulty is well recognized as being characteristic of this type of laryngo-tracheo-bronchitis. The response to the usual methods of treatment was decidedly poor and the oxygen tent served to

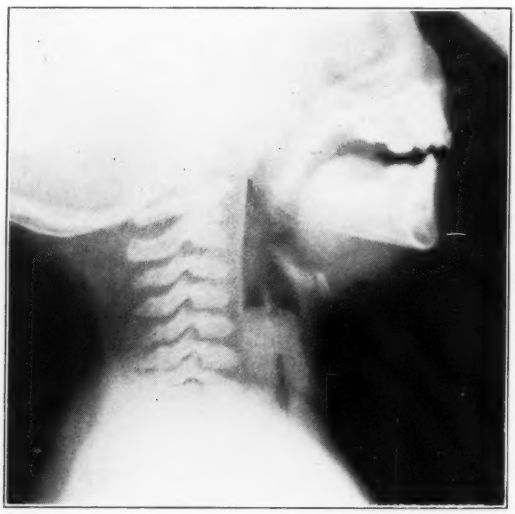

Fig. 2. Lateral view of the neck, showing marked edema of the arytenoids and marked peritracheal and periesophageal swelling.

make the treatment more difficult, in that it caused the mucoid discharge to quickly dry and thus rapidly increase the obstruction and dyspnea. Because of this increasing obstruction, the oxygen cannot be absorbed. The response of the bacteriophage instillation, together with the constant steamed air, on the other hand, produced unexpectedly striking relief of symptoms and was followed by rapid and complete recovery.

An unusual finding in this case was the presence of an abscess, low in the pretracheal region just above the superior mediastinum. Preoperatively the abscess was clearly seen by roentgen examination (Figs. 1 and 2). This is the first time the writer has encountered such a complication during the course of this disease. It was probably due to an infection and ultimate breaking down of the pretracheal and paratracheal lymph nodes which take lymph from the lower part of the larynx and trachea, the bronchi and bronchioles and communicate with the superior and the posterior mediastinal nodes and also the lower deep cervical glands.

CASE 2. K. C. B., a boy, aged one year, was admitted February 12, 1937, to the public ward service of Dr. H. Tobey at the Massachusetts Eye and Ear Infirmary. The child had had a head cold for several weeks. The day before admission, he developed a crowing inspiration with gradual increasing difficulty in breathing, but no cough nor any cyanosis. He had no history of foreign body or any such previous attacks.

Examination on admission showed a moderate amount of dyspnea with slight pulling present when the child was restless but much less when he was quiet. He was obviously markedly dehydrated and probably ketotic. The lungs revealed fairly good breath sounds, both sides being equal. No rales or prolonged expiration were heard. A laryngeal wheeze was transmitted symmetrically. There was no change in percussion. The heart was quite rapid; the larynx revealed marked congestion and slight edema of both arytenoids and ventricles. The cords could not be seen because of the large amount of sticky mucopus.

That same night, at 11:30 p. m., an emergency local tracheotomy was performed with marked improvement in breathing. A throat culture showed staphylococcus albus. In spite of the continued treatment, which included forced increase of fluid intake, several doses of both prontosil subcutaneously and prontylin orally, 10,000 units of diphtheria antitoxin, instillation of various fluids into the trachea in order to prevent formation of crusts, and tincture of benzoin steam tent, the child not only failed to improve but gradually acquired more and more difficulty in breathing.

Two days later, all previous medication, except the forcing of fluids and the steam tent was omitted, and ten drops of staphylococcus bacteriophage was administered through the tracheotomy tube every two or three hours. This was soon followed by a loosening of the cough, allowing the patient to raise easily large amounts of mucoid, and at times, bloody secretion through the tube, which was removed by suction.

The child did well for five days, when the temperature again rose to 103 degrees. The patient became drowsy and his body and face were covered with an erythematous urticarial rash which was diagnosed as a delayed serum reaction from the diphtheria antitoxin given six days previously. The following day, however, the urticarial wheals disappeared and the child's general condition improved.

A cork was placed in the tracheotomy tube the next day, that is, ten days after operation, but it caused restlessness and some dypsnea. It was therefore removed, but was again replaced two days later and allowed to remain for four hours without any reaction. The next day the cork was put in again and remained all day. The following morning, thirteen days after the tracheotomy, the tube was removed and a dry, sterile dressing applied. The child ate and drank well, had no dyspnea and very little cough. One week later he was discharged well to the out-patient department, where he has been seen several times since then and is in excellent general health.

Comment: This case also presents the typical course of staphylococcus laryngo-tracheo-bronchitis developing, however, after an upper respiratory tract infection of two week's duration. The patient continued to get worse in spite of every therapeutic effort, including diphtheria antitoxin and prontylin and prontosil. Improvement was not noted until bacteriophage therapy was used.

CASE 3. J. F., a girl, aged nine months, was admitted to the Massachusetts Eye and Ear Infirmary at 5:00 a. m. on the morning of November 2, 1935. The patient had had a head cold for the past week or ten days associated with nasal discharge and obstruction and a slight dry cough. For the past two or three days the child was extremely fussy, quite hoarse, coughed a great deal and had a temperature of 103 to 104 degrees. About 3:30 a. m. on the morning of admission, her breathing became rather difficult with gradual paleness and cyanosis. The pediatrician was called and found the child with marked dyspnea associated with retraction of the supraclavicular spaces and a temperature of 105 degrees. During examination of the throat, the patient suddenly ceased breathing for a few moments but soon recovered. She was rushed to the hospital, where a direct laryngoscopy revealed a very acute injection of the entire larynx, extending down into the tracheal mucosa. Both cords were also injected. No membrane, crusts or edema were present. There did not seem to be definite or ample cause for laryngeal obstruction, therefore it was deemed advisable to place the patient in a compound tincture of benzoin steam tent under careful surveillance by special nurses.

That afternoon the child suddenly became much worse, showing extreme labored breathing, marked supraclavicular and epigastric retraction and marked cyanosis. An immediate low emergency tracheotomy, under novocaine anesthesia was performed, and a small ring punch used to enlarge the tracheal opening. The tracheal mucosa was fiery red and presented a thick, velvety appearance throughout, but no secretion or crusts were present. A culture taken from the tracheal mucosa showed a pure hemolytic streptococcus.

Immediately after operation, the breathing became free and quiet and the child regained its normal color, but she appeared extremely toxic and dehydrated. She was replaced in the tent and her room was kept constantly well impregnated with benzoinated steam. No clyses were given, for the patient drank copious amounts of water and also received frequent rectal glucose taps.

X-rays of the chest showed narrowing of the larynx and no enlargement of the thymus. White blood count was 11,100, and a smear showed 72 per cent polymorphonuclear leucocytes.

On the seventh postoperative day the child seemed quite well. The tracheotomy tube was corked for a few minutes without any ill effects. This treatment was repeated daily, increasing the time each day, and on the eleventh postoperative day the tube was removed and the lips of the wound pulled together with strips of adhesive plaster.

On the fifteenth day the child was discharged from the hospital, the temperature, pulse and respirations normal, the voice normal and the wound practically healed.

Comment: This case illustrates the typical course of a streptococcus laryngo-tracheo-bronchitis, showing the following characteristics:

1. High febrile reaction, definitely higher than in the staphylococcus laryngo-tracheo-bronchitis.

2. Fiery red larynx, without any crusts or secretion on the cords.

3. Thick, red and velvety tracheal mucosa without any secretion or crusting.

4. Very marked toxicity and dehydration.

In this type, treatment by means of early tracheotomy, steam tent and general measures to overcome dehydration and toxicity usually is followed by a gratifying result.

DISCUSSION

From the cases here described, it is apparent that acute laryngo-tracheo-bronchitis is a real challenge to our profession. First, as regards its early diagnosis, and second, its management. Concerning the diagnosis of this condition, it is unfortunate that not infrequently it has been mistaken for such conditions as spasm of the larynx, laryngeal diphtheria, and in the late stages for broncho pneumonia.

Spasm of the larynx or croup may be easily distinguished by its very frequent history of previous attacks, its sudden onset usually at night, and the mildness of the inflammation which chiefly affects the parts above the cords where, on examination, there is usually found a very slight amount of congestion associated with a spasm of the muscles of the larynx. The attack lasts for four or five hours and slowly wears away and the child falls asleep. Most cases present simply a croupy cough, hoarseness and some inspiratory dyspnea, but rarely the associated expiratory dyspnea, together with the marked recession of the suprasternal and supraclavicular spaces and epigastrium encountered in laryngo-tracheo-bronchitis.

As regards laryngeal diphtheria, this disease can now safely be considered a rare condition. The onset may resemble that of laryngo-tracheo-bronchitis. However, nondiphtheritic laryngitis usually presents marked changes subglottically, whereas laryngeal diphtheria, although it may be primary in the larynx, is more commonly an extension from the pharynx. Direct smears from the laryngeal membrane, which should be taken in all cases, invariably reveal diphtheria bacilli. In such cases, even though antitoxin is administered on suspicion of diphtheria, it is imperative to supplement it by either an intubation or a tracheotomy. This is also extremely important in the treatment of laryngo-tracheo-bronchitis, and therefore, should be done early.

As regards the management or treatment of laryngo-tracheo-bronchitis, the measures that have been used have consisted of intubation, tracheotomy, frequently repeated bronchoscopies and suc-

tion through the tracheotomy tube, instillation of various fluids directly into the trachea with the hope of liquefying the dry crusts, injection of foreign proteins, forcing of fluids, oxygen tents and inhalations of steam.

These measures are undoubtedly all important. The writer is concerned, however, with those measures which are of the greatest value. For example, some have advocated that the patient be placed in an oxygen tent. This measure, in our experience at the Massachusetts Eye and Ear Infirmary, has proved valueless in this type of case, especially where staphylococcus is the infecting organism. In fact, it seems that it was a deterrant to active therapy, such as frequent suction, frequent forcing of fluids, etc. Furthermore, and most important, the cold air in the tent had a definite drying effect on the secretions in the tracheo-bronchial tree, thus greatly increasing the doctor's most serious problem. In our experience it has seemed of the utmost importance to place the patient in a croup tent and in a room small enough to allow keeping the air constantly moist. The patient should also be under constant supervision of a competent special nurse.

Another measure that has been resorted to, especially in early cases, is intubation. Although obstruction in the early cases may seem to be limited to the larynx and its immediate subglottic region, experience has shown that invariably the disease progresses rapidly into the trachea and bronchi. A mild or fulminating condition of this type of infection cannot be prognosticated at the beginning; hence it is advisable that tracheotomy should be performed as early as possible. The rationale for this is that since intubation usually proves insufficient, a tracheotomy invariably follows, therefore, the tracheotomy should be performed as soon as a diagnosis is made, so that valuable time will not be lost. This procedure will also greatly facilitate any necessary bronchoscopies, will allow for easy instillation of liquefying fluids into the trachea and bronchi, and will facilitate the frequent insertion of the suction tube by the indispensable attending special nurse.

There are only two conceivable objections to tracheotomy: first, that it leaves a scar in the neck, but this objection is certainly not a serious consideration in a condition where it is a question of saving the life of the patient; and second, that there might be a tendency for the ingrowth of granulation tissue into the tracheal lumen. This, however, has never been our experience at the Massachusetts Eye and Ear Infirmary.

The outstanding problem in the treatment of those cases due to staphylococcus infection, is to keep the dry, hard, semisolid secretions in the trachea and bronchi as liquid as possible, so that the patient may be able to cough them up. Since all other measures, such as high fluid intake, steamed tents, oxygen tents and various solutions, e.g., adrenalin, hydrogen peroxide, sodium bicarbonate and perborate, almost without exception have failed to accomplish this purpose in this type, the use of staphylococcus bacteriophage has been resorted to as described in Cases 1 and 2, with most unexpectedly good results. The use of bacteriophage in this type of case was first mentioned by Richards,[2] who made use of it in one of his patients who was given many other solutions without benefit. Since there have been no solutions reported that have proved of sufficient value in liquefying dried crusts, it would seem that staphylococcus bacteriophage is deserving of more thorough application. The action of bacteriophage is primarily liquefaction of staphylococcus. Its use, therefore has some rational basis, especially in view of the fact that in the staphylococcus type of case, death is most probably caused by the mechanical effect of the dry crusts leading to obstruction and the resultant exhaustion, rather than death by extreme toxemia, as in the streptococcus type of case.

As regards the streptococcus cases, the outstanding clinical manifestations are those of extreme toxicity, such as high fever, marked dehydration and striking necrosis of the tracheal and bronchial mucosa with pseudo-membranous formation. Dry crusting, so common in the staphylococcus type, is conspicuous by its absence. In these cases, therefore, bacteriophage therapy is not rational. The only measures available are those that have been mentioned, e.g., early tracheotomy and suction, a room constantly saturated with medicated steam, together with the usual general treatment of severe toxemia.

In all these cases roentgenograms should always be made for the possibility of a foreign body, and in order to pick up nonopaque foreign bodies it is of inestimable value that the roentgenograms should be supplemented by fluoroscopic examination of the chest by an expert.

SUMMARY

1. Three recovered cases of acute laryngo-tracheo-bronchitis, one due to hemolytic streptococcus and the other two due to staphylococcus albus, are reported in detail.

2. The present status of our knowledge of this clinical entity is discussed, and the important clinical differences between streptococcic and staphylococcic are stressed.

3. A plea is made for early diagnosis by pediatricians and general practitioners, as time is an extremely important factor.

4. In the treatment of cases of staphylococcus origin, staphylococcus bacteriophage proved of great value.

CONCLUSIONS

1. The proper handling of these cases requires that they be recognized early for what they are, and that valuable time should not be lost in treating them with antitoxin.

2. Immediately upon suspicion of the diagnosis the patient should be transferred to a hospital where direct laryngoscopy and bronchoscopy should be carried out at once and where more active treatment such as tracheotomy may be done at a moment's notice.

3. If time permits, prior to operation, roentgen study should be made; otherwise it should immediately follow the tracheotomy.

4. Other essentials in the treatment of these cases are:

a. Constant competent special nursing care.

b. A comparatively small-sized room in which the air can easily be kept well saturated, at all times, with steam containing tincture of benzoin.

c. A room equipped with a properly functioning suction apparatus.

d. Forcing fluids either by mouth, rectum or by clysis, especially in streptococcus cases.

e. In the staphylococcus cases, staphylococcus bacteriophage should be instilled into the trachea at frequent intervals, to be followed by immediate suction. Its use is urged in all such cases.

Appreciation is expressed to Dr. Harold G. Tobey for permission to report Case 2 from his service.

416 MARLBOROUGH STREET.

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XXIII

ELECTROCOAGULATION OF TONSILS

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PHILADELPHIA

Many physicans and otolaryngologists using electrocoagulation of tonsils today have been or are still pioneering in this field. These men, after trial and error, after conscientious study, thought and experimentation have adopted certain methods and technique which in their hands and judgment have given them safe and efficient results.

Many of those decrying this method have either given it scant study, or have witnessed someone not sufficiently versed in the procedure, and for these reasons have thrown it into discard.

In the electrocoagulation of tonsils, as in any other surgical procedure, the operator must be confident of his own ability, and this must be impressed upon his patient. He should have no hesitation in letting it be known that what he is about to do is not new, undetermined or experimental, but that the results obtained are definite.

Many of these patients are of the neurotic type and therefore choose this method because of a fear of surgery. Therefore, it is imperative to impress upon them the harmlessness of the method and the mild reactions that may follow treatment.

While electrocoagulation is as safe as any agent, it is nevertheless possible to cause undue reactions, and a proper knowledge and technique should avoid these.

Although I am a staunch advocate of electrocoagulation, I recognize that it has its definite advantages and disadvantages, and should not be used as a routine everyday procedure, but cases should be selected as in other surgical procedures. The more coagulations one performs, and the more one perfects his technique, the more nearly perfect are the results. I believe that anyone attempting to do this type of work should be well versed in the surgical removal and the anatomy of the tonsils.

Arguments have been advanced that electrocoagulation is time consuming for the patient. However, this is no valid argument.

The time element does not enter into cases where life may be endangered, or where morbidity may result from surgical removal. The time element is negligible in cases where electrocoagulation is definitely indicated. When one considers that a surgical removal incapacitates the patient from five days to a week, and perhaps longer if any complications arise, is it not more beneficial when no time is lost from work, no hospitalization and the aggregate time spent in coagulating the tonsils is far less than the time spent in the hospital? Is it not indeed a great sense of satisfaction in knowing that no harm has come to the patient and the end results measure up to, if they do not surpass, a surgical enucleation?

ANATOMY

Although the tonsils, after the age of 30, become progressively reduced in size and in old age are barely visible, yet some of the adult tonsils assume a very large size. It must not be forgotten that although the faucial tonsils become smaller, the lingual tonsils become more active, especially if the former have been removed. The free or exposed surface of the tonsils is covered by a closely adherent epithelial membrane which extends into the crypts, some of the crypts extend into the entire depth of the tonsil to the capsule. These crypts covered by the plica semilunaris above and the plica triangularis below, become filled with food, bacteria and debris and the tonsillar tissue in this area should be thoroughly eradicated. The existence of a definite capsule or membrane is questioned by some anatomists. The capsule is actually a separate layer, stripped from the adjacent pharyngeal aponeurosis. The latter encloses about four-fifths of the tonsil. The tonsil lies embedded in two muscles: the palatoglossus muscle or anterior pillar, which has a fan-shaped origin in the oral surface of the soft palate and terminates in the lateral aspect of the tongue, and the palatopharyngeus muscle forming the greater part of the tonsillar bed. The anterior pillar varies in size and thickness and covers the anterior half of the upper pole of the tonsil and a large part of the anterior and lateral surfaces. The posterior relationships of the tonsil are very important. It is here that we should be acquainted with what lies behind, so as not to injure any of these structures. Between the middle and superior constrictor muscles is a triangular space through which runs the stylopharyngeus muscle and the ninth or glossopharyngeal nerve. Opposite the lower pole of the tonsil, the ninth nerve runs forward, not far from the capsule, and is separated from it only by the palatopharyngeus muscle. The arterial and venous supply to the tonsil is rich and anastomotic. The internal carotid artery is about one inch

from the outer side of the tonsil, therefore wounding this vessel is not probable. The external carotid artery feeds the tonsil through five branches. Each vessel breaks up into small twigs on reaching the surface of the inner layer of capsule. The external carotid is still further removed from the tonsil than the internal, lying outside of the styloglossus and stylopharyngeus muscles. The two most important branches which may be injured are the ascending pharyngeal branch which is nearer the tonsil than either the main trunks, and the tonsillar branch of the facial artery. The facial artery itself, especially if it is tortuous or anomolous in its forward position, may be injured where it passes between the styloglossus and digastric muscles. The nerves that supply the tonsil and adjacent areas are the vagus, the lingual, the glossopharyngeal, and the sphenopalatine.

INDICATIONS

1. General medical conditions such as hypertension, endocarditis, nephritis, tuberculosis, or any other chronic pulmonary conditions and syphilis. In this class can be included any condition which would contraindicate active surgery.

2. Neurotics who are in need of tonsillectomy but will not submit to surgery. This embraces a large class who, because of fear of hospitalization, hemorrhage or other complications, absolutely refuse to submit to surgery.

3. Blood dyscrasias, such as hemophilia, moderate secondary anemia, and long bleeding and coagulation time.

4. Economy to the patient. There are many patients, especially during the recent period of economic stress and strain, who cannot undergo hospitalization and the outlay of an immediate sum.

5. Postoperative tonsillar tabs or remnants and small lymphoid growths.

6. Those above the age of surgical risk.

7. Lingual tonsil and varix.

8. Carcinoma[1] of the tonsil.

9. Public speakers and singers. There are no voice changes, as the vocal structures are left intact.

CONTRAINDICATIONS

1. Children. The ideal here is obtained by general anesthesia, plus any of the recognized methods of enucleation.

2. For those who would rather have the work done at one sitting by surgical methods.

3. Acute or recent infections of throat or tonsils. Outside of these I know of no contraindications for electrocoagulation of the tonsils.

ANESTHESIA

The anesthesia employed varies. Some use local anesthesia and others infiltrate. Maillard[2] injects a two per cent procaine and epinephrine 1/25,000 in the region of the superior fossa, infiltrating the capsule to a depth of about one inch. He claims this causes complete anesthesia of the tonsil, soft palate, and base of the tongue within five or ten minutes. The anesthesia is due to infiltration of the branches of the glossopharyngeal nerve which supplies this area. Although this should give good anesthesia, I cannot see why a patient should be subjected to repeated injections, with its dangers of infection and the possibility of injecting into one of the vessels. Silvers[3] uses pontocain two percent, swabbed on the pillars, pharynx and palate in hypersensitive individuals to allay the pharyngeal reflexes. Others[4] have used a 10 per cent cocain solution, swabbed on the tonsils and pillars every five minutes for three times. After extensive trials with various anesthetics, I have adopted the following procedure: The patient is instructed to take an anesthetic lozenge one-half hour before coming to the office. Another lozenge is taken fifteen minutes before treatment is begun. In hypersensitive patients with marked pharyngeal reflexes, the soft palate, uvula, pharynx and anterior and posterior pillars are painted with either a four percent cocain solution, pontocain two per cent solution, or one of the other anesthetic products. After waiting a few minutes, I swab the tonsillar area to be coagulated with equal parts of cocain and phenol by weight. This anesthetic is made by adding cocain and phenol crystals of equal weight to each other. This is slightly warmed by immersion of the bottle in warm water to facilitate liquefaction of the crystals. Immediate effect of swabbing the tonsil with this solution is a blanching of the mucous membrane. After waiting about ten minutes we are ready to proceed. After the first treatment, if the patient complains of a little more pain than I think the procedure should warrant, I attempt to cocainize the spheno-palatine ganglion. This has often helped me to get

the complete co-operation of the patient. In many cases the pain, at the time of the treatment, has been either nil or so insignificant that the patient offered no comment. I have not seen any reaction or any undue sensitivity to either cocain or phenol in any of the cases. I have not seen any burns, edema or sloughing following the use of this anesthetic. It has given me more complete anesthesia than anything else that I have tried. Of course, one must be careful not to use an over-abundance of the solution, to prevent it from trickling into the larynx. The phenol also acts as an escharotic aiding materially in reducing the tonsillar tissue. As a topical application I have also used diothane, five per cent solution. Apply twenty or thirty drops of the solution to all parts of the tonsil and pillars. Repeat this application two or three times at five minute intervals, then proceed with the electrocoagulation of the tonsil. This solution is of relatively low toxicity and causes no perceptible irritation when used in the manner suggested. Epinephrine or ephedrine should not be used in conjunction with this solution, as these drugs retard penetration and reduce the efficiency of the anesthetic. Another topical anesthetic which gives good results is Rorer's procain butyrate topical solution used in the same manner as above.

EQUIPMENT

The machine used should be one that has an even current, free from shock. The one that I use is the conventional long-wave H. G. Fischer model "H." This machine has a pre-adjustment unit which I usually set between 250 to 500 milliamperes, depending on the size and density of the tonsil. Any long-wave machine may be used which is not of too old a vintage. Good equipment is essential to satisfactory results. As a rule the cheaper machines do not produce the same current and are not as safe to use as the medium and higher priced ones. To follow this technique it is necessary to have an apparatus that will deliver an even and steady current and, above all, one of low voltage to eliminate entirely any possibility of sparking. The electrode is one devised by Haiman of New York.[5] Jaros of Chicago, and others have also developed a biactive tonsil coagulating electrode, constructed with two active curved needle electrodes which project one quarter of an inch from the insulated holder. The needles of the electrodes are set either two or four millimeters apart. Coagulation takes place only in the space between them. The large block tin indifferent electrode which has been heretofore employed is eliminated. This electrode consumes two-thirds less current, as the tissue resistance to be overcome between the needles is only two or four millimeters, as against five or six inches of flesh

and bone tissue resistance with the indifferent electrode applied to the neck. There have been no sloughs or burns with the biactive electrode when properly used. It is certainly possible to have a slough with the unipolar method. Although the biactive electrode is used wherever possible, yet we must revert to the unipolar method in the final fulgeration of the tonsil. Here we use either the unipolar ball electrode or the unipolar needle with the indifferent electrode attached to the patient's arm. The latter two electrodes mentioned are used to remove small remnants of the tonsil or to smooth the fossa of remaining tonsillar tissue. The tongue depressor used is the conventional type made from hard rubber. I use a modified Dillinger pillar retractor and tongue depressor combination made from hard rubber. There are several nonconducting pillar retractors and tongue depressors on the market which will answer the purpose satisfactorily. The important thing is to get accustomed to a certain set of instruments and use them efficiently.

PROCEDURE

A direct head lamp,[6] strongly illuminated, is more useful than the indirect reflected light. The patient is comfortably seated, and at the first visit it is noted whether the tonsils are of the large infantile lymphatic type, not adherent to the surrounding structures, or of the small fibrotic embedded type. The size of the anterior pillars, the depth of the oral cavity, and the amount of reflex gagging by touching the anterior pillars are noted. All these criteria point to either an easy or a difficult exterpation. A history is important to determine whether the tonsils have previously been removed, whether the patient has had repeated attacks of sore throat or quinsy, as the latter type requires a great deal of skill and judgment. It is also important to ascertain whether the patient has or has had any disease that would point to any focal infection. In this particular type of case, small fractional treatments over a longer period should be given because of the autogenous vaccine effect, rather than more massive treatments.

The treatment should not be one where the time element is a factor. Massive coagulation of the tonsils with a purpose of removing it in less than the number of treatments required will result in dissatisfacton because of pain and possible bleeding from sloughing. The pillars should not be coagulated if possible. Coagulation of the palatoglossus and palatopharyngeus muscles may result in pain and edema with subsequent fibrosis, making it difficult to remove the tonsils. Although I have seen no harm result from removing the

plica triangularis when it is very large and extends into the pharynx covering the tonsil, yet for a better cosmetic effect and for less distress to the patient, I usually leave it intact. Always be sure that the structures are well anesthetized before attempting treatment. Make sure that the connections on the machine, the amperage, and the instruments are all in working order. I anesthetize the structures as I have outlined under anesthesia. The patient is taught to depress the tongue, holding the tongue depressor in the right hand for the right tonsil, and in the left hand for the left tonsil. This keeps the patient's mind occupied, and allows the operator to use his hands freely and eliminates an assistant. The operator holds the electrode with his right hand while treating the right tonsil, and in the left when treating the left. The bipolar electrode is inserted into the tonsil usually beginning in the midsection. The prongs are directed away from the pillars and the foot switch applied for a few seconds. A whitish area of coagulation is produced between the two needle points. The electrode is applied several more times several millimeters apart. This leaves a studded appearance with uncoagulated tissue between the coagulated circles. If there is any gagging the electrode should be withdrawn and a few minutes allowed for rest before resuming. The number of treatments depends on the size and type of tonsils and the rapidity with which the patient wishes to have it done. The large boggy tonsil will be dehydrated more quickly. There is no danger in overcoagulating or hemorrhage at the beginning of treatment of this type of tonsil. It is the small fibrotic, embedded tonsil or the tonsillar tab which gives more trouble if overcoagulated or the coagulation extends beyond the capsule. Usually it has been my custom to treat one tonsil at each sitting, the sittings one week apart. The tonsil will usually be found from 25 to 50 per cent smaller after the first treatment. After the fifth treatment one is working in less tissue and nearer to the important structures. The depth of penetration of the needle and the amount of current now requires more acute judgment. The question often arises at this stage whether one is down to the capsule or whether it is muscle or tonsillar tissue. This same problem arrises in surgical removal also. Therefore the operator's experience enters here. However, by applying the cocain and phenol solution to the area in question, certain phenomena will aid us. The area, if tonsillar tissue, turns white and its surface is not smooth; if muscle or capsule is anesthetized, the area is grayish in color and is smooth. After a number of treatments, one can readily distinguish the tissue by the above appearances. After about nine-tenths of the tonsil is removed, we resort to the unipolar electrode, using an indifferent electrode. For small lymphoid follicles we use

the needle electrode, and for smoothing the fossa the ball electrode. Whenever in doubt about the tissue to be coagulated, whether it is muscle or tonsil, we use the ball electrode, as very little harm can be produced. After completion, we allow the patient a three or four weeks rest and have him return for a checkup. At this time the fossæ are explored for any remnants. The upper and lower poles are examined and the posterior area of the anterior pillars. The fossæ should be perfectly clean, comparable, if not better, than a surgical tonsillectomy. I have watched these patients over a period of time and have found no recurrence of tonsillar tissue.

During treatment, patients with focal infections have had their symptoms increase. Those with arthritis or kindred conditions have complained of pains in the joints, malaise and general constitutional disturbances. The absorption of the end-products of the tonsillar tissue by electrocoagulation has the same effect as using an autogenous or stock vaccine or a foreign protein substance. Silvers[3] states "the virulence of the invading organism determines the severity of the general responses or autogenous vaccine reaction. When the infection is either attenuated or eliminated, as is usual after the fourth treatment of each tonsil, there is rarely a general or grippe-like reaction. Fulgeration, though sealing the lymphatics and inhibiting the nonspecific protein reaction, does not interfere with the absorption of the end-products of bacterial destruction. It is the absorption, of necessity which accounts for the unabated vaccine reaction which affords the patient so much relief of both subjective and objective symptoms."

For one to be so dogmatic and state that pain can be eliminated entirely would be for one to state that pain does not exist. Pain differs in the type of individual, type of machine, type and amount of anesthesia, and the knowledge of the anatomy, physiology and technique of the operator. Pain is due to the amount of trauma to the normal structures. If only the tonsils are coagulated there is very little pain. However, if the normal surrounding structures are coagulated, not only will there be pain at the time of treatment, but afterwards. If the technique of anesthesia is followed as outlined there will be very little pain or discomfort. The important thing to remember is not to overcoagulate or do too much at each sitting.

Although hemorrhage is possible and can be very troublesome, I have seen only one case of slight bleeding and that was controlled very readily. I have heard of cases of bleeding, but I believe there

must have been some error in technique or lack of judgment. Naturally, if one coagulates too deeply or extensively, sloughing leaves the vessels open. If the bleeding does occur, the same means can be used as if it is encountered in surgical removal.

CONCLUSIONS

This is a safe and efficient method.

It does not and should not replace surgical removal, but is an alternative method in the removal of tonsils.

It is used when surgical removal is contraindicated.

A thorough knowledge of anatomy and surgical principles is prerequisite to the correct use of electrosurgical technique.

A method of anesthesia is outlined.

2123 PINE STREET.

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Clinical Notes

XXIV

ENCAPSULATED EMPYEMA OF THE PETROMASTOID: REPORT OF A CASE*

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On securing a cure of otitis media and severe mastoiditis without surgical intervention, the otologist usually feels that he has accomplished a difficult and praiseworthy feat and that further trouble from the mastoid without a new infection need not be considered a possibility. A series of cases encountered at The Mayo Clinic in the past four years has thrown considerable doubt on the proposition that one need not expect further trouble from a mastoiditis that apparently has been healed and has not caused symptoms for as long as a year.

Recently, Lillie and I submitted a paper for publication in which we reviewed six cases of encapsulated empyema of the petromastoid. The last three cases in that paper are cases of which I had the entire care, and they demonstrate serious intracranial symptoms from the lighting up of an apparently quiescent encapsulated empyema of the mastoid or petrous pyramid after a latent period of fifteen, ten and five years, respectively. I shall now present a similar case in which there occurred a latent period of slightly more than a year.

REPORT OF A CASE

CASE 1.—A woman, aged 37 years, came to the clinic and stated that three years previously she had had bilateral otitis media and mastoiditis and that a continuous discharge had occurred from both ears for about a month. She had then seemed completely well and continued so until a little more than a year later. She then began to have pain of gradually increasing severity situated back of the right

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eye, also projected from the right mastoid toward the vertex. About a month later diplopia developed and she began to have attacks of vertigo with a tendency to fall to the right.

Examination by her local physician was reported to show a choked disk "of several diopters and a rotary nystagmus with the rapid component to the left." Also she was found to have paralysis of the abducens nerve on the right side.

She was referred to a neurosurgeon, who performed a ventriculography and found the ventricles normal; he then performed a craniotomy in the right temporal fossa for the purpose of preserving the eyesight. This produced a slight remission of the pain and considerable relief from vertigo and diplopia. The retro-orbital pain soon began to occur in attacks of increasing severity.

She was admitted to The Mayo Clinic approximately three years after the beginning of the acute otitis. At that time her chief complaint was of severe paroxysms of lancinating pain situated for the most part deep in the right orbit. She was found to have choked disks of approximately one diopter, a paresis of the right external rectus muscle, grade 1 (on a basis of 1 to 4) and a rotary nystagmus to the left, grade 1. Caloric testing showed the re-activity of both labyrinths to be within normal limits. There was a slight degree of deafness on the right of the stapes, fixation type. A series of roentgenograms, both the base plate and the Stenver position, showed sclerosis of both petrous pyramids of an equal degree. The symptoms pointed generally to involvement of the anterior surface of the petrous pyramid, but in our experience choked disks were not found associated with petrositis, and it was not felt that the symptoms were sufficiently definite to warrant exploration, so the patient was asked to return for observation in a month.

On her return there had been a moderate increase in the severity of the retro-orbital pain as well as the vertigo and nystagmus. An encephalogram was made which was reported to demonstrate a chronic arachnoiditis which was thought sufficient to account for the papilledema. Therefore, exploratory operation on the right mastoid and petrous pyramid was advised.

At operation there was evidence of disease of the cells in the tegmen, and on removing them an epidural abscess involving the undersurface of the right temporal lobe was discovered. Although there was a rather suspicious looking downward tenting of a small

portion of the dura, I did not think that there were sufficient indications of an encephalitis to warrant an exploration of the temporal lobe at that time. Although intracranial extension into the temporal lobe might explain the vertigo and nystagmus, the typical retro-orbital pain would be difficult to explain on this basis. Perilabyrinthine cell tracts were therefore searched for above, posterior to and below the labyrinth, and although all three canals were skeletonized on their lateral aspects, a lead to the apex could not be discovered. A radical mastoidectomy was then resorted to, but a fistula could not be found in the hypotympanum or above or below the eustachian orifice. The cutaneous incision was therefore extended upward and downward in front of the auricle (the incision recommended by Eagleton) and the aponeurosis of the temporal muscle was incised. When an attempt was made to elevate the temporal muscle over the previous craniotomy, it was found firmly adherent and could not be elevated. Thus we were forced to do our exploration of the petrous pyramid in a considerably restricted field. The base of the zygoma was removed until we were able to throw the eustachian tube and semi-canal for the tensor tympani into one. The temporomandibular ligament was cut at its posterior and superior attachment, was freed from the glenoid fossa and the jaw was drawn forward. The internal carotid artery was then exposed by removing the tympanic bone and the anterior wall of the canal with a gouge. After uncovering the carotid artery I was surprised to find that, below, the internal carotid artery and jugular bulb were in contact and there was no free space below the cochlea. The carotid artery was drawn forward out of the carotid canal and the apex of the petrous pyramid was entered through the medial wall of the carotid canal. A relatively large abscess approximately two cm. in length and one cm. in diameter, containing creamy pus and infected granulation tissue was immediately entered. The contents were removed by curettage and the opening was enlarged so that a No. 14 French catheter could be placed down to the apex of the cavity and brought out through the aural canal. The usual plastic operation was done and the postauricular wound was closed.

The patient was completely free of retro-orbital pain for the first time in three years and she was dismissed from the hospital on the fifth postoperative day. A week after the operation there was a sudden return of retro-ocular pain, vertigo and nystagmus. It was found that the drainage tube had become plugged. On correcting this the symptoms immediately disappeared and the convalescence was otherwise entirely uneventful. Four months have

passed since the patient was dismissed from our care, and in a recent letter she reported that she is still free of symptoms.

Although an unpleasant emotion is engendered by the fact that complications may arise from an apparently healed mastoiditis from one to fifteen years after an apparent cure, the otologist should be aware of the possibility that such a complication may occur and alive to the possibility that petrositis may occur even though it may have been initiated by a disease apparently cured years previously.

MAYO CLINIC.

XXV

FIBRO-LIPOMA OF THE BRONCHUS: REPORT OF A CASE

THOMAS H. MCGLADE, M.D.

CAMDEN, N. J.

Benign tumors of the bronchus are no longer of sufficient rarity to warrant special interest in themselves. A survey of the literature, however, reveals the fact that only two tumors of this particular histological type have been reported as existing in the bronchial tree. The first case noted of this variety was by Myerson in 1928. The second was reported by Wessler and Rabin in 1932, and was included in a report of seventeen benign bronchial tumors. In a recent review of the literature, Pollack, Cohen and Gnassi, list only one fibro-lipoma in a hundred and four cases of primary bronchial neoplasm. Fibromata and lipomata were also relatively uncommon, with adenomata being by far the most common variety of benign tumors of the bronchus.

The symptomatology of bronchial tumors is readily understood, if one keeps in mind the all-important factor of bronchial obstruction. Early, when the tumor is small, irritative cough which is not specifically related to this condition is present and may be the only symptom. As enlargement of the tumor gradually takes place, the bronchial lumen becomes more encroached upon with a resultant interference with the inflow and outflow of air. The mechanical condition produces a wheeze, the intensity of which is directly dependent upon the degree of bronchial obstruction present. It is important to constantly keep this fact in mind, viz., that wheeze is the most important characteristic symptom of bronchial tumors and that wheezing patients should not be classified as asthmatics, until careful study has definitely ruled out the presence of bronchial neoplasms.

If such tumors are not recognized in the early stages and removal accomplished, permanent damage to the lung is bound to ensue. Complete obstruction of the bronchus causes atelectasis by absorption of the entrapped air. Later, because of inadequate drainage, infection occurs distal to the obstruction, with bronchiectasis and pulmonary suppuration being the end results. This suppuration distal to the tumor will eventually take the life of the patient unless

drainage is established. Even complete removal of the tumor at this time will not result in a return to normal of the damaged lung. Bronchiectatic dilatations, if present, will always persist. It is this course of events that led Morlock and Pinchin to state that benign bronchial tumors are benign in the histological sense only.

REPORT OF A CASE

CASE 1.—F. H. B., aged 48 years, male, white. He was admitted to Cooper Hospital on January 31, 1938, complaining of cough and expectoration, pain and rattle (wheeze) in left chest. His present illness began five weeks ago, with pneumonia, from which he never fully recovered. His personal history revealed that he had had three attacks of pneumonia during the past year and that he had lost forty pounds in weight. Physical examination was negative except for the chest, which showed diminished expansion to be present over the left lower lobe, with tenderness to palpation and dullness to percussion, over the same region. No rales were heard. All laboratory studies were also negative, including three sputum examinations, for tubercle bacilli. The roentgenogram of the chest showed the left lower lobe to be rather hazy. The mediastinum was displaced to the left. The picture was that of left lower lobe atelectasis.

Bronchoscopy was performed on February 7, 1938, and the following conditions were found: The larynx, trachea and right bronchus appeared normal. The left bronchus was almost completely occluded by a rounded tumor mass which extended to within a few centimeters of the carina. Its point of attachment could not be determined. This mass appeared smooth and not ulcerated. The bronchial wall exhibited no evidences of fixation or rigidity. The condition was thought to be a benign neoplasm.

On February 12, 1938, the patient was referred to Jefferson Hospital, Philadelphia, Pennsylvania, where the obstructing tumor (Fig. 1) was removed by Dr. Louis H. Clerf.

The pathological report submitted by Dr. Baster L. Crawford, pathologist of Jefferson Hospital, was as follows: The specimen consisted of an elongated, polypoid mass, which measured 5x1¾ cm. The external surface was smooth and pink in color. The mass was solid and composed of tough grayish-yellow tissue. Histologically the specimen was composed largely of fibrous tissue and fat. Some areas were partially covered by a transitional mucosa. No gland structure was observed in any of the sections. The diagnosis was: Fibro-lipoma of the bronchus.

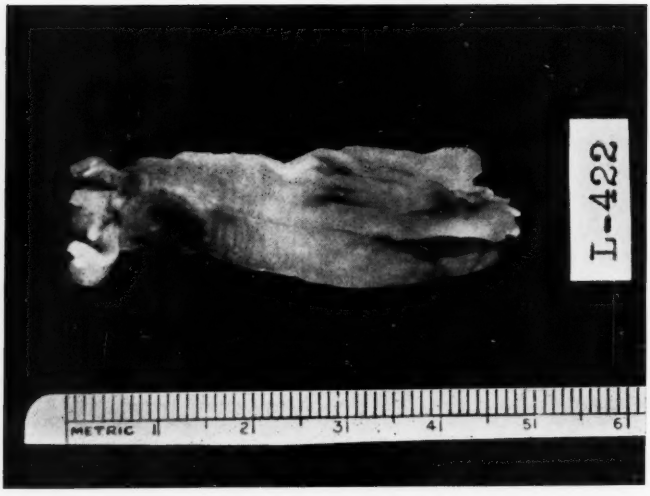

Fig. 1. Fibrolipoma removed bronchoscopically from left main bronchus.

Immediately following removal of the tumor, the patient was asked to take a deep breath, which he did, with great relief and satisfaction, adding the comment that he had been unable to do so for over a year.

A follow-up bronchoscopic examination was performed on February 24, 1938. The left main bronchus was found to be patent and the former site of attachment completely healed. A small amount of mucoid secretion was aspirated from the left lower lobe subdivisions. The patient was instructed to return to the clinic at a later date. At this time we had planned to inject iodized oil into the left lower lobe to determine the presence of bronchiectasis. Unfortunately he did not return, but it is assumed that moderate bronchial dilatations were present.

COMMENT

Bronchial obstruction is the pathological entity responsible for the symptom, wheeze. Allergic conditions causing spasmodic contractions of the bronchi are most commonly the cause of this symp-

tom. However, one must not assume that such is the condition at all times. Bronchoscopic examination will definitely decide if the case is not clear cut. In the case of benign bronchial tumors, removal of the obstructing mass is urgent, because delay will result in permanent damage to the lung.

SUMMARY

A fibro-lipoma of the bronchus is reported. The diagnosis was made and apparent cure accomplished by bronchoscopy.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, October 3, 1938

THE PRESIDENT, DR. THOMAS J. LEWIS, IN THE CHAIR

Early Diagnosis of Laryngeal Malignancy

FRANCIS L. LEDERER

(Author's Abstract)

My presentation this evening is purely informal and therefore unpremeditated. Any virtue it may possess, and all its shortcomings, arise from the fact that it is given without deliberation. My method of approach may subject me to scorn of those who will feel with incredulous rage that I fail to dwell long enough on those favorite avenues which they feel are all-important. That is inevitable, but I hope that they will consider that I offer but an individual's impression, without bias, guided by the experience which it has been my good fortune to have—well realizing that I am addressing myself to those whose ideas are rather set; who feel complacently capable of following a pattern. The danger of this, as every exponent of scientific medicine knows, is retardation of progress. When every physician who has an interest in the subject of carcinoma of the larynx, and even malignancy in general, realizes that the problem cannot be solved by taking sides, then we shall have advanced a long way. Any group unwilling to accept this philosophy, in itself constitutes an economic and social cancer. I say these things because I feel so keenly that scientifically and commercially one modality has been pitted against another.

One of the most intriguing studies in medicine is the differentiation of disease entities which have a similarity of symptoms. Hoarseness is one of those symptoms; by this is not implied a loss of voice (aphonia) but any interference with the intensity, pitch and quality of voice tone.

Hoarseness may be defined as a roughness or a discordance in the quality of a voice influenced either by an alteration of pitch or intensity, or both. There are numerous anomalies which are manifest at birth. As man progresses through life, there is perhaps no organ, by virtue of its position and function, which is subject to greater mistreatment or abuse. Diseases of the upper and lower respiratory tract, vocal abuse, systemic disease, diseases of the surroundings of the larynx and external irritants are largely responsible for many abnormal manifestations. The causes of such changes are numerous and should be known not alone to the laryngologist, but to the general practitioner, who must be made to appreciate the importance of voice changes, and to the layman, who must be made to realize that this symptom is a danger signal of the laryngeal dysfunction ("voice and hoarseness conscious").

It must be borne in mind constantly, however, that hoarseness and inspection do not always enable the establishment of a diagnosis objectively. The laryngeal mirror must replace the guesswork which is so commonly employed in the diagnosis of hoarseness. It is too often assumed that hoarseness is merely due to an irritant source, and a regime of voice rest and tincture of benzoin inhalations will clear up the condition. No one would attempt to make a diagnosis of a pulmonary affection without properly auscultating and percussing a chest, or conclude that there was a kidney condition without a urinalysis or renal functional test. A complaint referable to the larynx deserves the same approach, viz.:

(1) A careful complete case history.

(2) Indirect mirror laryngoscopy.

(3) Complete general physical examination, particularly of the upper respiratory tract.

(4) Roentgenologic studies of the sinuses, chest and neck.

(5) Blood examination, both microscopic and serologic.

(6) Repeated examination of sputum.

(7) Direct laryngoscopy, bronchoscopy and esophagoscopy.

(8) Biopsy.

As a laryngologist progresses through his span of professional activity, he comes to realize more and more that every cause of con-

tinued hoarseness must be considered in arriving at a proper diagnosis. While he may tentatively feel that a certain condition exists, it is his desire to rule out by elimination those diseases which present analagous laryngeal pictures. He therefore considers the classification of chronic laryngitis:

1. Chronic non-specific laryngitis.
   - (A) Chronic hypertrophic (diffuse and circumscribed).
     - (a) simple
     - (b) chorditis nodusa
     - (c) pachydermia
     - (d) hemorrhagic
     - (e) subglottic
     - (f) contact ulcer
   - (B) Laryngitis sicca.
   - (C) Perichondritis and arthritis.
2. Specific laryngitis.
   - (a) tuberculosis
   - (b) syphilis
   - (c) scleroma
3. Neoplastic.
   - (a) benign
   - (b) malignant.
4. Paralysis.
   - (a) functional
   - (b) organic

In a purely didactic approach to the cause of laryngeal change, there is a tendency to enumerate points in favor of each condition with an unjustifiable abandon. It is only by careful elimination of all possibilities that an absolute diagnosis is reached.

Hoarseness which is pronounced, persistent and progressive, arouses the suspicion of carcinoma. Inspection by indirect and direct laryngoscopy enables the laryngologist to study the lesion for diagnosis and to rationalize treatment. It should be borne in mind that the pathologic changes of the larynx lie in more than meets the eye. An infiltrative lesion is not always established by immobility of the vocal cord.

Experienced laryngologists have a wholesome respect for the site of the lesion as of prognostic significance. A difference in the

distribution of lymphatics is responsible for the fact that a carcinoma on the true cord, or below it, may exist for a long period without manifesting metastatic qualities. No so with the lesions above this area or those on the cord close to the arytenoids, when metastases are common. The choice of therapy is often dependent upon such deductions.

A word about the type of malignancy is not amiss. Let it be understood that for all practical purposes the site of the lesion and what effect it is having upon the host (both local and general) are more important than the cellular structure. In my experience the removal of tissue for histologic study is imperative and is not attended by danger of spreading the disease. Removal of tissue by indirect laryngoscopy whenever possible is advised, and all tissue removed should be sectioned. Numerous bits of tissue are removed at times, and it is necessary to study all, in order that malignancy which may be absent in some, may not be overlooked. Excision should be thorough, particularly in depth.

Intralaryngeal procedures of any kind, except in the very incipient stage, are rejected because they are ineffective, and therefore dangerous. For any operable malignant growth, radical surgery augmented by electrocoagulation is the only justifiable measure. Laryngofissure preliminary to the removal of the cord, electrodissection or electrocoagulation, have proved satisfactory, at least in selected cases of early involvement of the anterior two-thirds of the cord. The chances for recovery are absolutely nil when at an early stage thorough removal of the growth cannot be effected. When such procedure can be carried out satisfactorily, the prognosis becomes relatively favorable.

The safest rule for the management of laryngeal carcinoma is surgical intervention whenever the case is seen near its incipiency, and nonsurgical if seen in an advanced stage. If, on careful examination, there appears to be no peripheral involvement and the growth is so situated that it can be, so to speak, punched out with considerable surrounding tissue by special cutting forceps, this may be resorted to under direct laryngoscopy or by laryngofissure. An ulceration or thickening near or at the edge of a cord may thus be removed. Unfortunately, such cases in clinical practice are comparatively rare, the laryngologist usually being consulted when the process has already advanced too far for this comparatively simple procedure. Complete extirpation of the larynx then is the only procedure capable of affording some chance of recovery. Laryngec-

tomy is now advocated by most experienced laryngologists when the limits of the false cords are not passed; that is, when only the tissues of the true cord proper are involved and when there is no glandular involvement.

Treatment of carcinoma of the larynx is medical or surgical. The management of laryngeal carcinoma by means of irradiation has not been as uniformly successful as surgery. We have diligently followed through case after case with the accepted mode of therapy and find ourselves woefully lacking in encouraging statistics. We are awaiting the time when this form of therapy emerges from its stage of epiricism to produce such results that we can have complete confidence in its employment.

It is perhaps unnecessary to state that an important chapter has been added to the annals of surgical advance by the progress which has been made in the treatment of laryngeal carcinoma. No more need the tremendous morbidity of the operation be feared, for hopelessness is not justified with a procedure, albeit formidable, which contains the promise of a permanent cure. Such results, formerly the experience of but few, are attributable to early diagnosis made possible by better training of physicians, an improved armamentarium, laboratory facilities for diagnosis, and the widespread education of the public to regard pronounced, persistent and progressive hoarseness with suspicion. The operation itself has not been greatly changed in the past decade, but selection of the patient through meticulous examination by the internist and proper preoperative preparation have greatly enhanced its therapeutic result. Newer methods of rectal anesthesia and the more general use of local analgesia also have materially aided in reducing the danger of the operation which formerly incurred a great mortality.

Rehabilitation of the patient's voice is of utmost importance. Whenever removal of the larynx is suggested for carcinoma the immediate question is, "What about the voice?" because the possible loss of speech is by no means a pleasant thought. The temptation to reply that "dead men do not talk" is suppressed in favor of the humane retort, "Yes, but why place speech foremost, when life itself is endangered? Besides, with a tumor in the larynx, there is no normal or even audible voice." This in a sense of fairness to one who has been apprised of a dread disease and cannot think logically in the first shock.

While life is always placed uppermost in the attack of the cancer problem, we must nevertheless be in a position to rehabilitate the

patient, building up what has been torn down by the conquest of disease. To this end, the patient is informed that it is possible to speak without a larynx through the development of a pseudo-speech or by the use of an artificial larynx. The latter is a simple device which allows the patient soon to resume his social and business contacts. A number of patients who have undergone laryngectomy begin quite early to exhibit a form of speech, producing consonants with the buccal muscles, bringing the air in the mouth under a certain pressure with the articulatory narrowing. The vowels themselves are heard weakly as appendages to the preceding consonant, using the aftersound of the consonantal murmur. In this type of speech respiration does not enter into play.

Living Laryngectomies

(slides and photographs)

JOSEPH C. BECK

The loss of one's voice, however hoarse or however difficult it is to speak, is a most important consideration to the patient when you propose removal of the larynx. I have found this to be the greatest obstacle and have tried to fight it by showing phonograph records and movietones of laryngectomized patients, and by having them talk to other patients who have had a laryngectomy and learned to talk with an artificial larynx. Yet they were not sold on the operation. I see I am discussing Dr. Lederer's paper rather than my subject, so I had better get busy on my own topic.

The material I am presenting is not a group of walking delegates, such as Dr. Lederer showed, but is a series of photographs, only four of them, selected over a period of years from the time I started to do this work in 1905 until 1935. Since that time this major surgery has been done by my partner, Dr. M. R. Guttman. Those who have seen laryngectomies know that great progress has been made and there should be a great many more living laryngectomies than those resulting from my operations. When I first started this work the two outstanding complications were mediastinitis and pneumonia. Then came a period of conservatism during which we performed very few laryngectomies, but rather laryngotomies, followed by x-ray treatment in about the same dosage the dermatologist would use today for a superficial skin lesion. Then came MacKenty's monumental work. From then on we began to benefit from laryngeal resection.

The four pictures shown are examples which I wish to describe specifically. Statistically, I have performed 212 laryngectomies in private practice for cancer of the larynx. Of these, 37 remained well without recurrence for more than five years. Those of the 37 that succumbed, did so through intercurrent disease, and wherever it was possible to obtain a postmortem there was no metastasis demonstrable. Of the 37, fourteen are still living, and it is with a great deal of satisfaction that I say in spite of the unemployment situation today, twelve of the fourteen are earning their own living. I think they are certainly entitled to a pension!

CASE 1.—G. F., a man, aged 78, of Superior, Wisconsin, had a diagnosis of squamous cell carcinoma of the larynx. He was operated upon (laryngofissure) by a general surgeon in one of the largest clinics in the northwest, in 1920. The surgeon declared him inoperable for laryngectomy, and he was advised to return home for palliative treatment. Two years later he was referred to me by his family physician because of great difficulty in breathing and a hurried tracheotomy was necessary. At his behest and insistence I performed a radical resection of the larynx in December, 1921, including tributory glands of the neck and a greater portion of the contiguous esophagus. This patient had a very stormy time following operation, in that he had two attacks of erysipelas which extended all the way to his scalp and downward to the abdomen. I wish to emphasize these erysipelas attacks in view of the subsequent history of nonrecurrence. This patient has been and still is active as sheriff of his town.

CASE 2.—B. K., a man, aged 76, of Chicago. Biopsy showed squamous cell carcinoma of the larynx. Laryngectomy was performed November 1, 1928, in the usual manner, and subsequent examination of the diseased parts of the larynx showed that there also was a fibrotic type of tuberculosis present. The patient never had any lung symptoms of tuberculosis. He made an uneventful recovery. He has had two attacks of osteal tuberculosis with recovery. This patient, like the first one, was never able to use an artificial larynx but depended upon the "burping" type of speech.

CASE 3.—H. S., a man, aged 61, of Milwaukee, had a diagnosis of squamous cell carcinoma. The usual laryngectomy was performed in March, 1925, with complete recovery. The unusual part of this case was the fact that neither the bucco-esophageal voice or the artificial larynx satisfied him, and he conceived the idea of improving his speech by poking a hole, with the aid of a red-hot ice pick, from

the upper part of the tracheal ostium to the back of the tongue. He kept this opening patent by persistent use of a goose quill for several weeks so that the tract never did close. He has learned to close this opening at meal times so that food and drink do not escape into the lower respiratory tract. By means of his finger placed over the tracheal ostium he is able to force sufficient air through and thus produce a more even voice, in fact, the best voice I have ever heard in a laryngectomized patient.

CASE 4.—Doctor B., a man, aged 64, of Chicago. This case is interesting from three standpoints: first, that two varieties of carcinoma were diagnosed; second, the apparent futility of the use of the Coutard treatment and, third, the use of a Levine feeding tube instead of a gastrostomy, which the patient refused. Seven years prior to his second visit to my office, I saw the doctor with a growth on the right ventricular band, and advised biopsy. When I saw him seven years later, he had been tracheotomized in the interim and now was unable to swallow even water. There was a great mass of carcinomatous material in the esophagus and larynx. As stated above, he refused gastrostomy and, following a marcelment resection of the larynx and esophagus, performed by Dr. Guttman in February, 1939, a Levine feeding tube was inserted. The patient made an uneventful recovery and existed on this type of feeding for seven months, when he developed a sudden cardiac failure and died. The two types of carcinoma diagnosed were transitional cell, in which the Coutard type of treatment was used, and correctly so, but it failed, and secondly, the mass we removed showed a squamous cell carcinoma. Apparently there was no other metastasis about the body, although we were unable to obtain a postmortem.

DISCUSSION

(Papers of Drs. Lederer and Beck)

DR. THEODORE J. WACHOWSKI: I came here with no misconception, because I understand Dr. Beck's attitude on the subject, and I am glad that Dr. Lederer has prepared the way for the apologia which I came to give but which I consider unnecessary. I would remind you of the early days of laryngeal surgery, as reviewed by Douglas Quick, when the nail on the forefinger was the scalpel and the gruesome first attempt at laryngectomy in New York was remarkable because of the fortitude of the patient.

I remind you that radiology is only 40 years old and that Coutard therapy is only 18 years old. Radiation biology is so young

that there is still disagreement on some of the important facts, and only recently have we learned to measure x-ray and radium on a comparable basis.

If you consider the multiplicity of factors in radiation therapy, the necessity for checking the role of each of them individually, the confusion created by the rapid improvement in equipment, and then realize that even the five-year cure period is not entirely satisfactory, it must be apparent that the evaluation of our statistics is time consuming. This explains our delay in reaching the definite conclusions you demand. I would like to review the situation briefly.

Regaud noted the differential action of gamma rays on the various cell types and Coutard applied this finding to the therapy of upper respiratory tract malignancies. He found that a beneficial dose had to be close to the limit of tolerance of the tumor bed and that such a dose could not be delivered in less than fifteen days without risk of serious damage. While such a short course was satisfactory for radiosensitive tumors, a longer, less intensive course appeared to be more desirable for more differentiated cells, apparently due to the preservation of the status of the vasculo-connective tissue. Prolongation beyond 60 to 90 days, however, might result in radiovaccination of the tumor.

Large treatment portals reduced the amount of therapy that could be given because of toxemia and the danger of late radiation damage. Most of the successfully treated cases fell within a certain dosage zone, beyond which cures were rarely effected, while the danger of radiation damage was increased.

While the biopsy specimen may give a clew as to the radiosensitivity of a lesion, the location of a laryngeal tumor has been the best index as to probable curability. Roughly, the chance of cure decreases as the tumor spreads, or is primarily located, away from the epithelium of the vocal cords.

Some statistics have been published, the best of which are Coutard's 20 to 25 per cent five-year cures. There is no way of comparing surgical and radiologic statistics, however, because the surgeon chooses only the favorable cases, while practically all cases are treated, at least for palliation, by the radiologist. We get late cases at the Research and Educational Hospitals, and our statistics are poor. In order to present any statistics of cure, the patient should be followed to the postmortem table, to be certain that there were no carcinomatous rests.

I think it is entirely justifiable to do what Dr. Brunner tells me was done in Vienna a few years ago. They decided they would give Coutard therapy a trial, and so treated all their cases of carcinoma of the larynx by that method. That does not mean that they were convinced of radiation's superiority—some were absolutely opposed. But the five years will be completed in a short time and they can say that irradiation had done this or that, and their statements will have the weight of adequate trial.

DR. MAX CUTLER: The question of surgery and radiation in the treatment of carcinomas of the larynx can be divided into three parts:

1. Large, extensive, inoperable, extrinsic carcinomas of the larynx which are outside the domain of surgery but which, as a rule, are composed of highly undifferentiated cells and are radiosensitive. This group of cases requires no discussion, as they are admittedly inoperable both technically and biologically.

2. At the other extreme, we encounter small carcinomas limited to the true vocal cord, which are technically operable. When the microscopic examination of the biopsy discloses an adult hornifying squamous type, and if the lesion has not reached the anterior commissure, surgical removal gives excellent results and is the method of choice. There exists, however, about 20 per cent of lesions which affect the true vocal cord and which are technically operable but biologically inoperable. In spite of their small size and technical operability, they are composed of undifferentiated cells which have extended along the lymphatics anteriorly to the commissure or posteriorly to the arytenoid. Surgical removal of these lesions is commonly followed by local recurrence, and the surgeon is surprised to find a local recurrence resulting from the removal of a lesion which seemed so favorable and so definitely operable. This group is radiosensitive and responds well to radiation therapy. It would be interesting if the surgical statistics segregated this group and an attempt were made to determine whether the failures are not composed of tumors exhibiting this histologic structure. Perhaps this 20 per cent group would yield better results if the lesion belonging to it were treated by radiation. There are no radiation statistics, however, to support this thesis at present.

There exists an intermediate group in which a decision between surgery and radiation is difficult. This difficulty may be due to the lesion being of borderline operability, or to the patient's age or his general condition which may contraindicate operation. In this group

the individual factors in each case must be carefully considered. These factors are histologic and clinical. Histologically, if the tumor is highly undifferentiated, it is generally radiosensitive. If it is of an adult squamous form, it is generally radioresistant. A more accurate guide to radiosensitivity, however, is the fixation or mobility of the cord. The highly undifferentiated tumors are almost without exception movable even when they are extensive; the differentiated types are nearly always fixed. When a discrepancy exists between the histologic structure and the mobility of the parts, the latter is the more reliable sign.

It is important to recognize that fixation may not be due to neoplasia alone but may be due to inflammation. A moderate amount of radiation invariably results in a re-establishment of mobility when fixation is due to inflammation. This response may serve as an accurate and important guide in determining this point and deciding between surgery and radiation in borderline cases.

DR. L. B. BERNHEIMER: My own experience with radiation therapy of laryngeal carcinoma has been much more fortunate than Dr. Lederer's. I have seen at least twelve five-year cures of intrinsic laryngeal carcinoma, and one extrinsic who is living and well for five years without signs of disease.

At Hines Hospital we have attained Dr. Lederer's conception of the ideal relationship between the radiologist and the surgeon. Each patient with laryngeal carcinoma is studied both by Dr. Cutler from a radiologic point of view and by myself from a surgical one before a decision is made as to the type of therapy to be employed. We have certain biologic and anatomic principles which guide us in formulating our opinions. The biologic considerations have been indicated both by Dr. Cutler and by Dr. Wachowski, who quoted Coutard. The anatomic considerations briefly are as follows:

1. All primary subglottic malignancies are treated surgically. Incidentally, the number of primary subglottic lesions seen is much greater than we heretofore believed.

2. Infiltrating lesions resulting in a fixation of cords are usually treated surgically. The therapeutic test of 2,000 roentgen units has, however, influenced our decisions. I have seen fixed cords become movable following such a regime, in which event radiation is continued. When such a cord becomes movable, we feel that the fixation resulted not from actual infiltration of the malignancy itself, but from inflammatory changes due to secondary infection.

3. Extrinsic lesions are usually radiated. I have seen an extrinsic lesion respond to radiation to the extent that only the intrinsic primary growth remained. This larynx has then been considered operable and has been subjected to the indicated surgical procedure.

4. Intrinsic, noninfiltrating lesions are usually radiated, especially those whose biology indicates radiosensitivity.

Sometimes, however, factors which are beyond our control determine the types of therapy. Such a factor is an intercurrent upper respiratory infection during a course of radiation. Such an infection may cause marked local reactions, necessitating the substitution of surgical for radiologic therapy. We have also seen patients who refused surgery. These patients have preferred to chance radiation rather than submit to laryngeal surgery, even after it was explained that in their particular instance surgery offered a better chance for cure.

DR. FRANCIS L. LEDERER (closing): Since I talked on early carcinoma, naturally I left out many things like lateral pharyngotomy and the like which are applicable in extrinsic lesions.

I wish to call attention to a case Dr. MacKenty published in the Archives in 1929, a case that went from 1922 to 1927 through the gamut of every type of diagnosis before the growth assumed a most characteristic appearance. We could all study that case very carefully and profit thereby, because it typifies what often happens. I did not mention x-ray pre- and post-operatively. We do employ it post-operatively to get any stray cells that may be present. I would warn you about leaving a permanent rubber feeding tube in situ for a long period of time. I remember a case published in the Journal of the A. M. A. as a cured laryngeal case following radium. The destruction of tissue which followed irradiation was so great that the man had to be fed by nasal catheter. It is interesting to note that we found positive evidence of malignancy despite the tremendous irradiation.

Radiosensitivity must be qualified. Today I saw a patient with laryngeal involvement who responded beautifully to irradiation, only to suffer from recurrence. One should bear in mind that radiosensitive tumors can return to become radioresistant and offer a tremendous problem so far as future treatment is concerned. Subglottic carcinoma is of great interest and occurs more frequently than we realize. In the French literature there is a percentage strikingly high; I believe 25 per cent of carcinoma of the larynx was

subglottic. In addition, carcinoma occurring in the laryngeal ventricle should have our attention, because for a time the diagnosis may not be established until studied by direct laryngoscopy.

I am pleased to hear Dr. Cutler's discussion, yet I believe it is irrelevant so far as my discussion was concerned. Many points have been advanced this evening, and I believe had Dr. Cutler been here he would have taken an entirely different approach. It is absolutely worthwhile to have a laryngologist working with the irradiation specialist. It would be interesting to have five-year cures presented in person, with laryngeal pictures and biopsies. However, if all the statistics are as bad as ours, especially with reference to follow up, I wonder if some of the 18 cured cases reported by Dr. Bernheimer are not some that visited our clinic or yours with recurrences. If these are cured cases, even over a five-year period, we must doff our hats to him and consider that something worthwhile is being done for laryngeal carcinoma and that the future promises much for radium and x-ray. However, so long as I can promise more certain results with surgery, I shall continue to advise surgery in laryngeal cancer until the other modalities can supply better statistics.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 7, 1938

THE PRESIDENT, DR. THOMAS J. LEWIS, IN THE CHAIR

Some Experiences With a Vital Stain on Otolaryngological Tissues

ELLISON L. ROSS

(This paper appears in full on page 212 of this issue.)

DISCUSSION

DR. J. R. LINDSAY: I have listened with interest to Dr. Ross' paper, which gives us further results of his investigations on the ear, and think he is to be congratulated in following up this line of work. The evidence presented here that mercurochrome will pass through the epithelium and mucosa of the middle ear, or the mucosa of the nose or sinuses, and then through the bony walls of the frontal sinus and the overlying dura in such a short space of time is at least surprising. The question which obviously arises and which Dr. Ross has been trying to answer is: how does the drug pass through? Is this a matter of simple diffusion? Or does the extension proceed to the deeper parts, by way of lymphatics or capillaries?

Dr. Ross has done former experiments, placing various substances, both crystalline and colloidal, in the middle ear and studying the effect upon labyrinth function.

We have also used some of these substances and have been unable to explain why some substances such as strong salt solution in the middle ear produce deafness and some other substances which we have used do not cause deafness, e.g., glucose solution, plaster of Paris, when sufficiently diluted that it does not become solid. The whole question of chemical changes which occur in the internal ear, and also of physical change, has rather successfully resisted investigation.

It is known that some crystalline substances will not pass through living membranes; for example, phenosulphophthalein injected into the sac of a hydrocele does not pass through the walls. It would seem that in the case of mercurochrome the action is that of a simple diffusion, in which the substance is not confined to lymph or blood vessels but spreads through all tissue. The rapid diffuse

spread of the drug throughout the mucosa and even the bony walls might raise the question again of the therapeutic value of this drug as an antiseptic. However, the fact that the drug diffuses into the tissues cannot be taken as evidence that it could be present there in sufficient concentration to have therapeutic value, or that it would not have a detrimental effect, for example, on the action of the cilia. It is true that chemotherapy has received a boost in recent years on the discovery of the action of sulfanilamide, but from the evidence to date the beneficial effects obtained by chemotherapy seem to be limited to a few drugs, most of which apparently have a specific effect on certain types of organisms.

DR. GEORGE T. JORDAN: I would like to ask the method of instillation. Was the mercurochrome put through the tympanic membrane with a hypodermic, or merely dropped onto the drum?

DR. SAMUEL J. PEARLMAN: The penetration of the mercurochrome through the mucosa and bone into the dura is to me an astonishing thing. There is one thing not quite clear to me. If I understand it correctly, the mercurochrome is placed in the nose for a certain time, the animal is perfused with a mild acid solution and this was carried on for several hours to several days. After a while the animal had in its vessels and tissues only a dilute acid solution. The question is, is it possible that some of the drug was dissolved and carried in through the bone and to the dura by the blood vessels and deposited where it was found? I should like to ask if control experiments were made? Were the tissues sectioned after staining and without the perfusion? It is easy to see mercurochrome in the tissues, and how can one tell that the remarkable depth of penetration was not due to the methods used?

DR. ELLISON ROSS (closing): Dr. Jordan has asked how the compound was applied. A blunt needle was put through the ear drum and the mercurochrome was injected into the middle ear.

Dr. Pearlman has asked if the perfusion might not have carried the stain in the tissues. The fact that the depth of the penetration of the tissues was the same in cases of varying thoroughness of perfusion, argues against the stain being carried by the injected solution. Mercurochrome seemed to have passed into the tissues much like diffusion. The part played by the circulation is not yet determined. It was hoped that knowing the mode of travel of mercurochrome in tissues we might be aided in the use of therapeutic agents that are intended for deep tissues.

Thank you for the discussions.

Inflammatory Diseases of Meninges and Brain of Pharyngeal Origin

HANS BRUNNER

(Author's Abstract)

The author described three cases, in one of which the base of the skull had been examined thoroughly macroscopically and microscopically. From these studies the following conclusions were drawn:

1. In the course of a pharyngogenous sepsis the meninges and brain may be affected by two means: (a) by means of a phlegmon of the parapharyngeal space; (b) by means of a general sepsis.

2. Infection of the parapharyngeal space may occur, with a critical reduction of temperature and increasing leucocytosis, but without alarming general or local symptoms.

3. Neuralgias of the trigeminal nerve can be absent in cases of phlegmon of the parapharyngeal space.

4. The locking of the eustachian tube in phlegmon of the parapharyngeal space occurs through infection passing along the connective tissue and along the blood vessels, which perforate the cartilage of the tube under normal circumstances from the parapharyngeal space into the mucous membrane.

5. At operation of deep abscesses of the neck, a normal sheath of the blood vessels should not be incised when there is a purulent necrosis in the surrounding fascia of the neck.

6. When infection of the parapharyngeal space reaches the contents of the skull, first the meninges become infected, then intradural suppuration, thrombophlebitis of the cavernous sinus and of the carotid plexus develop. All these diseases of the meninges are, as a rule, accompanied by typical, familiar symptoms, but sometimes these symptoms may be missed. Contrary to those infections produced by way of a phelgmon of the parapharyngeal space, the general sepsis first affects the brain itself, while the infection of the meninges does not occur or is of secondary importance.

7. The infection of the brain in general sepsis may lead to various pathologic entities. In these cases edema of the brain was produced.

8. This septic edema of the brain may appear clinically as a disease localized in the posterior fossa.

9. The septic edema of the brain may disappear with recession of the pharyngogenous sepsis.

DISCUSSION

DR. SAMUEL J. PEARLMAN: To say that I enjoyed this paper would be putting it mildly. Apart from the exceedingly interesting subject, the vivacious manner and enthusiasm with which Professor Brunner gave this presentation, gave me a great deal of pleasure.

We have known for a long time that parapharyngeal abscesses have a tendency to travel downward into the mediastinum. You all know that it is necessary to drain the neck in these cases and use the tamponade of Marshik to prevent descent into the mediastinum. However, I am certain that from time to time the literature will present cases of parapharngeal abscess, and will call our attention just as forcibly to the reverse situation; that is, that the process may travel upward to the base of the skull and kill by meningitis. It should surprise no one if a patient who has tonsillitis, then peritonsillar abscess, followed by a parapharyngeal abscess, should die of meningitis. I should like to again trace some of the pathways which have already been mentioned, as much for my own benefit as yours. One asks himself, how does the infection travel, and the answer is that there are a number of pathways which should be obvious to anyone who considers the anatomy involved. Pus deep in the neck goes as high as the base of the skull. It is only one step to go through the basilar foramena, particularly the foramen ovale and spinosum and lacerum. The infection may attack the bone and produce meningitis by direct extension. That would be a long-standing process and is a theoretical consideration only. By the time the bone at the base of the skull is involved enough to involve the meninges, the patient would probably be dead or recovered from his parapharyngeal infection.

There is another method in which the brain may be involved, and here are the pathways as I know them: It is possible for the retrotonsillar veins to become involved; from here to the jugular vein and the jugular bulb, thence to the cavernous sinus by way of the inferior petrosal sinus. This is not common, but does occur and death takes place by meningitis from the cavernous sinus involvement. Or, from the retrotonsillar veins there is a spread to the pterygoid plexus; once this is involved the cranial cavity is attacked by veins which go through the foramen spinosum, lacerum and foramen ovale to the cavernous sinus; or the spread may be by way of thrombophlebitis from the tonsils to the pterygoid plexus, through a deep communicating vein, then to the anterior facial vein, to the angular vein, and finally through the orbit by way of the ophthalmic veins to the cavernous sinus.

There is another way, i.e., directly by way of the walls of the carotid artery. Ira Frank reported infection in the carotid artery walls up to and into the brain. Still another route is by way of the plexus of veins which surround the carotid artery. These become involved from the pus which bathes the carotid artery or from a thrombosed internal jugular vein which communicates with the carotid venous plexus. The cavernous sinus is then attacked by extension through veins leaving the foramen lacerum.

One should not be surprised that in this patient the usual signs of cavernous sinus thrombosis were absent. Eagleton, a long time ago, called attention to the fact that the cavernous sinus may be involved from in front, medially and from behind. The fixation of the bulb and the orbital cellulitis which we associate with classical cavernous sinus thrombosis is seen chiefly in those cases where the sinus is involved from in front by way of the orbit. So that if the cavernous sinus is involved from behind the orbit, you cannot expect to find fixation and protrusion of the bulb until very late.

There is one other point which should be brought out in these carotid arteries. Not only meningitis can occur, but brain abscess. The infected clot in the carotid artery may be loosened and then lodge in the brain to produce an abscess.

I should like to thank Dr. Brunner for his fine presentation. The time is coming, and this is one of the occasions, when the profession will become increasingly aware of the fact that parapharyngeal infection travels upward as well as downward and can kill by invading the brain just as it does by going downward to produce the mediastinitis.

DR. SHERMAN SHAPIRO: I think it may be superfluous, but perhaps I have some reason especially to express my admiration for the masterly manner in which this first case report was presented.

Some of you may recall that several years ago I presented a study of parapharyngeal infection following tonsillectomy. At that time I combed the literature very thoroughly but never found anything like this, tracing the pathways of infection. I do not know why some infections travel upward and not downward. I do not know whether anyone can answer that. Among the cases I reported there were three fatalities, one from meningitis. No postmortems were obtained. It is evident that the percentage of cases who die from brain complications is considerably smaller, so that the more common route is apparently downward.

I was interested especially in Dr. Brunner's point about edema of the brain. Within the last two years I have seen two cases, referred by the neurosurgical department, with pontine posterior fossa symptoms; one had hemiplegia, a child aged about 12. Both cases were seen long after the initial focus, in one case a mild otitis which passed off, and in the other an upper respiratory infection. The initial focus had already passed and we were dealing strictly with the intracranial phase of the situation. I never thought of edema of the brain; I thought there was an area of encephalitis, as mentioned by Dr. Brunner. The second patient was recovering and the hemiplegia was subsiding. The other patient was sent home in poor condition and I do not know the outcome. It is certainly worth while to think of edema.

Another condition to consider is arachnoiditis. In any involvement of the posterior fossa one must think of circumscribed and other collections of cerebrospinal fluid, which may contribute to brain tumor.

I can only thank Dr. Brunner once more for the privilege of seeing this excellent presentation.

Dr. Noah Schoolman: I rise to take part in this discussion with the thought that fifteen years ago I had the privilege of listening to the lectures of Professor Brunner in late Vienna, but had no opportunity to talk back, and two years ago my son had the same privilege. So I take the liberty now to emphasize the close relationship of the temporal bone to the pharynx. Aside from the fact that the middle ear communicates with the pharynx by way of the eustachian tube, the apex of the petrosa as it enters the angle between the basilar process of the occipital bone and the body of the sphenoid, invades the pharynx to a point anterior to the plane of the pharyngeal tubercle, and thus becomes a part of the bony framework of the pharynx. Under these circumstances it is conceivable that a petrosal abscess may point into the pharynx and may be drained through that route. Some of the pharyngeal abscesses of obscure nature reported in the older literature as of otitic origin may have been of this source. It is, therefore, of interest to reflect that the presence of an abscess in the pharynx of uncertain etiology, exhibiting intracranial manifestation, may be a sinking abscess of intracranial origin, or a pharyngeal abscess which has extended upward. The difference would be important in the treatment—if the former, one would drain through the pharynx and hope for the best; if the latter, one would be led to intervene intracranially and expect the worse.

DR. HANS BRUNNER (closing): I thank you very much for your interest in my paper, and particularly Dr. Pearlman for his interesting views about the ascending infection.

Infections of the meninges from parapharyngeal abscesses are dangerous, because even when we find one and can open it, it is practically impossible to eliminate all pathways to the skull and its contents, since the ascending infection travels as a rule by more than one pathway. That is the first point to be emphasized. The second is that in these microscopically proven cases, they must be carefully examined, for when one finds pus in the carotid sheath or elsewhere, it is difficult to determine whether it is an infection traveling from below upward or from above downward. Therefore the interpretation of the microscopic picture is difficult. The third point is that those cases who die of what is thought to be a parapharyngeal abscess with descending infection, who have not corresponding findings in the mediastinum at autopsy, should be further examined especially with regard to the brain, for it is surprising how often the cavernous sinus will be found to be involved without having produced clinical symptoms of such a process.

Dr. Shapiro spoke of cases with general increase of brain pressure and local symptoms in the posterior fossa, often thought to be encephalitis but which, in reality, is often nothing more than edema of the brain. It is well to remember that generalized brain hypertension may appear to be a localized disease in the posterior fossa, as my cases have shown.

I also wish to thank Dr. Schoolman for his interesting remarks.

Intrinsic Carcinoma of the Larynx: A New Instrument Modeled After the Jackson Bronchoscope for the Intralaryngeal Application of Radon

FRANK E. SIMPSON

(Abstract)

This demonstration of a new instrument for intralaryngeal irradiation was given to create interest among laryngologists who have the opportunity of seeing cases of cancer of the larynx more frequently than other practitioners, and also to induce others to try this method. The instrument itself was shown, the technique of administration described, and a motion picture exhibited of treatment of the patient whose case was reported.

This woman, aged 80 years, became hoarse in October, 1937. In March, 1938, when she could hardly speak above a whisper, a diag-

nosis of squamous cell carcinoma, Grade II, was made. The lesion was on the anterior third of the right vocal cord. Both cords were freely moveable. Radium treatment was begun on April 7, 1938; between that date and May 5, 1938, 5000 mc hours were given to the cutaneous surface over the larynx with the radium bomb at 4 cm. distance. On May 1, 1938, the patient's singing and speaking voice was restored and has remained unimpaired. The vocal cords appeared normal and have remained so up to this time. A phonographic record of the voice made on October 6, 1938, revealed a normal voice free of huskiness.

The author pointed out the following advantages of the intralaryngeal method described:

1. It is non-operative, a desirable feature in elderly and feeble patients.

2. No pain is experienced.

3. The patient's breathing is free and unobstructed during treatment.

4. The radon is held at a fixed distance—5 mm.—from the surface of the tumor, so that dosage is accurate.

5. Danger of injury to the larynx is minimized.

6. If irradiation fails, operation can still be resorted to if advisable.

DISCUSSION

DR. JOSEPH C. BECK: The old saying—the proof of the pudding is the eating thereof—is apropos here. These records show the patient after treatment. What we should have had was a record of her voice before treatment was begun so that we could actually know. We have the word of the operator that the patient was hoarse, perhaps not as hoarse as I am now, after a laryngitis for about ten days.

The lesion was a Grade II. We have heard that the grading in treatment with x-ray and radium really does not amount to so much after all. There have been cases that responded to deep therapy, both x-ray and radium, but the holding of the radon or any instrument for this length of time would be rather difficult in some cases. Yet I see where this work surely can be enlarged upon, that one could anesthetize the larynx by nerve block of the superior laryngeal, and have no great difficulty in holding the instrument. Having suction to get rid of any secretions that might accumulate is of additional help.

We are still being told definitely that with a Grade I or Grade II lesion of the larynx, that is, an intrinsic laryngeal lesion, it is an operative case only—because you are dealing with cancer. Yet in time only will this splendid work that has been shown to us tonight be verified; time will show just how good this treatment is. But that a laryngologist, as Dr. Hagens is, is working along these lines, gives me a great deal of confidence that the work may become of distinct value.

When I read the program I was prepared for a different discussion. I thought Dr. Simpson was going to tell us how he would get rid of us as laryngologists by the employment of a direct laryngoscope. This has been a wonderful discussion, and we should all be very happy that we have seen and heard it.

DR. THOMAS C. GALLOWAY: We have seen carcinoma treated with relatively small doses, with beautiful immediate results indicating a cure by radium, and I think that later disappointments in such cases have made some of us a little skeptical about some reports. We are all, I think, open-minded about the results we get in carcinoma of the larynx with it, but the proof is in the cures for five years or longer, and I think until we get such results we must suspend judgment.

It would seem to me that by this method not a very adequate dosage is applied to the tumor, especially when one considers that the radium might not be absolutely accurately placed in relation to the lesion. We know, of course, that radiation short of cure is likely to make a cancer resistant to later radiation, and may carry the patient past the time when other treatment is possible, but it certainly is a valuable contribution which can be further developed. Dr. Hagens could perhaps devise some way whereby the tube could be curved or angulated so that it might be tolerated for a longer time, and in some benign lesions such as multiple papilloma of the larynx, I think it might be extremely valuable, and I hope it will be.

DR. FRANCIS LEDERER: I do not think we can pass the subject without thinking of a former member of the Society, Dr. Otto Freer. I think we all recall Dr. Freer's enthusiasm for the direct application of radium to the larynx. I believe Dr. Beck will remember one case in which he made freehand drawings of the larynx to show the tumor disappearing under direct irradiation, only to have the lesion recur after a length of time; also, with no evidence of local lesion, but with metastatic extension. Therefore I do not think Dr. Simpson intends, nor should we accept the thought, that he is representing

this as a cured patient. He is merely bringing before us a new instrument which is certain to be advantageous. Of course in an 81-year-old patient it is very dangerous, because one would not want to entertain operative intervention in such a patient, and if it offers the advantages pointed out, I believe it is a worthwhile instrument to follow through.

Relative to its being held in place for ten minutes, I recall the times I try to hold even a laryngoscope in place to let five or six students look through it, and I know how I fail to hold it accurately for even the length of time since the first student saw it. I am wondering if Dr. Galloway is not right in that it might be difficult to hold the instrument exactly in place with respect to the lesion, especially if there is respiratory motion of the vocal cords which might alter the direction of the irradiation and one would have to change it from time to time.

With respect to the dosage, while it is not high, I think it has the advantage of being direct and not through tissue. I would assume the dosage might be thought of as sufficient, inasmuch as there is no filtration other than that provided by Dr. Simpson.

I appreciate the opportunity of seeing again that the radiologist has the co-operation of the laryngologist in the treatment of his cases.

Dr. Paul Hollinger: This method of radon application is extremely interesting. I think Dr. Simpson has given us a real advance in the treatment of these lesions and the method should have further application in diseases of the bronchi and esophagus. The ease of application of radon in this manner would suggest the possibility that we might depend on radon instead of surgery in certain cases. The choice in this particular case is extremely fortunate, since the age of the patient rather contraindicates surgery, although statistics still favor the surgical removal of intrinsic lesions.

Dr. Walter Theobald: I would like to ask a question which might be in the form of a suggestion. In screening, is it possible that the irradiation might be too great for the unaffected side, and in such a case could you screen one-half of the tube so that the entire dosage would be directed to the affected side?

Dr. Frank E. Simpson (closing): There have been so many points brought up that it will be difficult to discuss them all.

We have thought about additional screening for the normal side of the larynx. This is not feasible, partly because of the necessity

of air space for breathing. Moreover, 1 mm. of gold screens off 99.9 per cent of the hard beta rays, and even an additional millimeter would screen off but a very small percentage of those that are left.

So far as dosage is concerned—it seems small and could doubtless be increased. We were extremely cautious so as not to hurt the larynx, and really gave less than the toleration dose, knowing that if the dose proved insufficient we could add to it. Our dosage proved to be sufficient, however, as the tumor disappeared.

I was associated with Dr. O. T. Freer a good many years ago and helped him treat his cases. Recently, in looking up our records I found we treated 19 cases. The subsequent history of these cases I do not know, because they were Dr. Freer's patients and his death prevented a proper follow up. I do know, however, that while they did well temporarily, some had necrosis of the cartilage and terminated fatally. We made the mistake in those days of trying to give too much irradiation at one time, sometimes as high as 800 millicuries in the larynx for thirty minutes—i.e., 400 millicurie hours. Moreover, the instrument used at that time did not hold the radon at the proper distance from the tumor. I do not believe that a ten-minute exposure tends to favor metastasis. We use a large quantity of radon for a short period of time so that there is a minimum of traumatism.

We did not use the suction apparatus because of the possibility of disturbing the carcinoma and the morphine and atrophine administered prior to treatment seemed to check the secretions sufficiently.

We made the gold capsule containing the radon 4.5 cm. long so as to allow for a certain play up or down. If you take three points, one opposite the middle of the first tube, one between the two tubes and one opposite the middle of the second tube, and these three points be equidistant from the gold capsule and in a line 5 mm. from and parallel with the gold capsule, the radiation received at these three points is practically uniform. The outside surface of the instrument is marked in centimeters so that the distance from the upper teeth to the larynx can be noted. The operator inserts the instrument until the distal end is at the tumor, and then pushes the instrument down 2 cm. more, which brings the center of the gold capsule opposite the tumor. The vocal cords fix the instrument at the lower end and the operator has little difficulty in holding it mobile for the short time required for treatment.

Thank you very much for the opportunity of presenting this subject to the Society and for your interesting discussions.

Abstracts of Current Articles.

NOSE

Meningitis from the Sphenoid Sinus.

Teed, R. Wallace (Ann Arbor, Mich.). Arch. Otolaryng., 28:589 (Oct.), 1938.

The literature is reviewed of all cases of meningitis from the sphenoid sinus and presented along with abstracts of these cases. The author found the sphenoid sinus involved in about 15 per cent of all cases of sinusitis, and found infection of this sinus to be responsible for approximately 35 per cent of all rhinological-intracranial complications.

It is thought that the spread of the infection to the meninges is by vascular channels and secondarily through the vascular marrow spaces of the sphenoid bone.

One hundred and twenty-nine cases of meningitis from the sphenoid sinus have been collected from the literature. From this group there were found seven cases of extradural abscesses, seven cerebral abscesses and one cerebellar abscess, six suppurative and one hemorrhagic encephalitis, and forty-seven cases of purulent thrombosis of the cavernous sinus. In four cases there was a thrombosis of the superior longitudinal sinus and in two the internal carotid thrombosed within the diseased cavernous sinus.

Much is made of the relation between disease of the sphenoid sinus and insanity, with evidence that in certain cases resolution of the infection brought about relief of the mental aberration. Hope is expressed by the author that the treatment of these cases of serious involvement will be more radically and successfully treated in the future.

TOBEY.

Histology of the Nasal Mucosa in Experimental Anaphylaxis. (Histologische Untersuchung der Nasenschleimhaut bei Experimenteller Anaphyloxic.)

Andersen, H. C. (Copenhagen). Acta Oto-Lar., 26:5-562 (Sept.-Oct.), 1938.

Guinea pigs previously sensitized by intraperitoneal injection of horse serum were thrown into anaphylactic shock by repeated injection or by dropping into the nose, and the tissues were secured half an hour later. Three factors are immediately evident—

sudden, marked hyperemia; increased capillary permeability with considerable amounts of secretion, red and eosinophile cells in the tissue spaces and nasal cavity; and connective tissue edema of the submucosa and of the supporting cells in the olfactory region. These three factors come on instantaneously, as opposed to the moderate hyperemia shown with surface irritation by tobacco or by aqueous irrigation, or the venous congestion with white cells following silver nitrate solution. Andersen ascribes these changes to a colloid-chemical change in the blood plasma or to damage of the capillary epithelium.

FENTON.

LARYNX

Benign Tumors of the Larynx.

New, Gordon B., and Erich, John B. (Rochester, Minn.). Arch. Otolaryng., 28:841 (Dec.), 1938.

A review of 722 cases of benign tumors of the larynx seen at Mayo Clinic in the past thirty years is reported. At this clinic it has been found that benign tumors are not nearly so frequent as malignant tumors of the larynx. Using these 722 cases, the authors discuss the incidence, location, symptoms, diagnosis and treatment of these tumors. There are many cases presented to illustrate each type of tumor.

TOBEY.

Indications for Different Types of Treatment of Malignant Disease of the Larynx.

Imperatori, Charles J. (New York). Arch. Otolaryng., 28:585 (Oct.), 1938.

This article takes into account the history, examination, diagnosis and the surgical and irradiation therapy of cancer of the larynx.

"Cancer of the larynx is a serious disease, for it is rarely discovered early, regardless of all the 'shouting from the housetops' of laryngologists to their medical colleagues who first come in contact with the patient. Whatever treatment is applied in the individual case must be early in order to secure a measurable degree of success."

TOBEY.

Some Clinical Aspects of Vocal Cord Inaction.

Tilley, Herbert (London). J. Laryng., and Otol., 53:355 (June), 1938.

In this article are presented eighteen cases of impaired function of one or both vocal cords, in most of which the etiology is obscure

unless one wishes to ascribe the loss of function to a toxic neuritis of the recurrent laryngeal nerve. In several cases there was ankylosis of the crico-arytenoid articulation due to arthritis, gout and syphilis. Many of the cases are transient vocal cord paralyses accompanying acute infections and trauma.

The author is impressed by the frequency of impairment of function in the left vocal cord, whether this impairment be due to ankylosis of the crico-arytenoid articulation or neuritis, and feels that the left cord is more vulnerable than the right. In speaking with Sir Frederick Hobday, F.R.C.V.S., the author found this to be universally true in horses, which rarely have aortic aneurysms.

The author asks whether the longer course of the left recurrent laryngeal nerve may make it more susceptible to toxins and chemical poisons as well as mechanical injury or whether the left-sided body structures may be congenitally less resistant than those on the right.

DEAN, JR.

ESOPHAGUS, TRACHEA AND BRONCHUS

A New Device for Radium Application in Esophageal Malignancy: Radiology.

Rubenfeld, Sidney, and Schneider, Theodore. Radiology, 31:5 (Nov.), 1938.

Squamous celled carcinoma is the most frequent histologic type of esophageal malignancy and rarely yields distant metastasis. The authors present a review of the various applicators devised for radium tube and radon seeds application to carcinoma of the esophagus and submit a description, drawings and photographs of their applicator and technique of application. "Diagnosis in our minds, is satisfactorily established by roentgen-ray alone." The levels of the lesion are indicated topographically on the skin of the chest. Adequate morphine and atropine, cocainization of the nasopharynx, abstinence from food for four hours constitute the preparation of the patient. The position of the radium applicator is checked by the fluoroscope.

The radium dosage is 20 to 30 mg. radium, screened with one mm. platinum, one mm. hard rubber, in situ for 24 to 48 hour periods, repeated at three-day intervals until a dose of 4,000 to 5,000 mg. hours is given. Three cases are reported.

JORSTAD.

EAR

Suppuration of the Petrous Pyramid: When and How to Operate: Report of Thirty Cases.

Moorhead, Robert L., and Baker, John P. (Brooklyn). Arch. Otolaryng., 28:497 (Oct.), 1938.

This is a paper based on thirty cases in which stress is laid not only on the usual symptoms, but also on that of pain around the eye and in the frontal region, if present with other symptoms of petrositis. Other symptoms mentioned of importance are night pain, and pain in and around the teeth.

The operative treatment advocated consists of a very complete simple mastoid, followed by an exposure of the middle and posterior fossa dura with the removal of as much bone as is necessary to reach the labyrinth. Also, if necessary from this point, the dura may be elevated with exposure of the anterior portion of the pyramid. By removal of the solid angle an approach is also offered for tapping the basal cisterns to reduce pressure and thus give better exposure. By this procedure the authors avoid a radical mastoid and thus fulfill two objectives: first, to save the patient's life, and second, to save the function of the ear. All objections to this procedure are didactic rather than practical.

Of the thirty cases thus treated, twenty recovered, and in most cases fairly good hearing remained. In no case was a radical mastoid necessary.

TOBEY.

Diagnosis and Differential Diagnosis of Deafness.

Crowe, Samuel J. (Baltimore). Arch. Otolaryn., 28:663 (Nov.), 1938.

The differential diagnosis of the various types of deafness cannot be made with tuning fork or audiometer tests alone. The tuning forks must supplement the audiometer if the test is to be of any value. The differential diagnosis can only be made after taking a detailed history and after examination of the upper air passages, the tympanic membranes and the eustachian tubes. The use of some form of masking apparatus on the good ear is absolutely essential in all hearing tests. In conclusion, the author feels that a sound knowledge of the anatomy, physiology and pathology of the part is very essential for the understanding of functional disorders.

TOBEY.

Pseudobotryomycosis of the Auricle. (Su due casi di pasudobotriomicosi a localizzazione rara (padiglione auricolare).

Cavazzani, F. (Padova). Arch. Ital. di Otol., Rino., Laring., 50:39 (January), 1938.

This equine disease is not very common in man, and when it occurs, it is usually located on the internal surface of the hands, in between the fingers, on the upper lip, or on the scalp.

Cavazzani reports two cases in which the condition manifested itself in the auditory concha, and states that in the literature he has been able to find only one similar case, which was reported in 1927 from Citelli's Clinics.

In man, this condition is characterized by a painless, pedunculated, single growth of about the size and appearance of a mulberry, dark red in color, and bleeds easily. The epidermal surface is macerated and covered by a serous secretion. It prevails in youth.

Histologically it resembles exuberant granulation tissue, the stroma of which is rich in connective tissue and is excavated by numerous vascular lacunæ. Some of these cavities are lined by a single layer of endothelial cells and others are surrounded by proliferating reticular elements. There is also a marked infiltration of lymphocytes and plasma cells and a scarcity of elastic fibers. The microscopic appearance offers the reason to dispute the correct terminology for this sickness. The author thinks that the term of telangiactatic granuloma, used by the Americans, should be adopted.

The etiology of this malady has been attributed to many microorganisms, such as the amoeba, mycocytes, etc.; but the staphylococcus aureus is the bacterium constantly found in the lesion.

Pseudobotryomycosis follows light trauma, small open wounds and friction. It usually develops on unclean individuals.

The permanent cure consists of complete excision of the peduncle deep into the skin and cauterization of the wound with pure silver or electric current.

SCIARRETTA.

Sudden and Temporary Deafness Due to Cochlear Congestion. (Improvvisa e passeggera sordità da congestione cocleare.)

Zanzucchi, G. (Milano). Arch. Ital. di Otol., Rino., Laring., 50:24 (January), 1938.

A night watchman, 45 years old, while on duty, became suddenly deaf and a very loud buzzing developed in both ears. No

evidence of head cold, acute inflammation in nasopharynx or middle ears was present. The vestibular apparatus reacted normally to rotatory and caloric tests. The patient was afflicted by articular rheumatism in the right shoulder which had been intermittent for the past two years. A very small percentage of hearing was present when tested with tuning forks and voice. Diagnosis of rheumatic congestion of the cochlear portion of the internal ear was made. The patient was hospitalized and immediately treated by injection of iodides alternating with actylcholine. His hearing returned almost to normal and tinnitus disappeared in approximately two weeks.

This malady was first described in 1926, and later in 1933, by Citelli, who has treated about 10 cases of this type of deafness.

This clinical entity is characterized by a sudden and marked unilateral deafness accompanied by very loud tinnitus, without middle ear involvement or vestibular disturbances, and usually occurs in elderly individuals with rheumatic diathesis.

It is to be differentiated from Ménière, tuberculosis, basal meningitis, traumatic labyrinthitis, purpura hemorrhagica, etc. Usually early and adequate energetic treatment procures excellent results.

SCIARRETTA.

Cancer of the Middle Ear and Cholesteatoma: (Épithéliomas de l'oreille moyenne et cholestéatome).

Cabarrony, J. E. (Bordeaux). Rev. de L. O. R., 58:722 (Sept.-Oct.), 1938.

This extensive study (42 pages) of cases in which epithelioma developed following mastoidectomy for the removal of cholesteatoma seems to suggest that the operative procedure may act as the exciting agent in bringing about the growth of cancer, or may reveal an incipient carcinomatous situation started by the pressure of the cholesteatomatous mass. For this reason Cabarrony and Portmann advise careful removal of every bit of the cholesteatomatous matrix, and tissue examination of any suspicious granulations springing up in the cavity.

FENTON.

Mastoditis Involving the Zygoma; Pathology and Clinical Course. (Zur Pathologie und Klinik der Zygomatikomastoiditis.)

Gadolin, H. R. (Viipuri, Finland.) Acta Oto-Lar., 26:5-492 (Sept.-Oct.), 1938.

Seventy-four of 957 cases of mastoiditis showed cells in the zygoma, mainly in well-pneumatized bones, but occasionally where arrest of pneumatization had occurred in the mastoid proper. Thirty-

nine of these cases showed the following special symptoms: Pain in the temple; swelling and tenderness of the temple and cheek, swollen eyelids; occasionally painful mastication; sagging of the upper anterior wall of the meatus. Meningeal irritation shown by increased cell-count and pressure was frequent; there were thirteen epidural abscesses, three brain abscesses and four cases of apicitis with epidural involvement. The other thirty-five cases exhibited only the usual mastoid symptoms.

Since intracranial penetration is frequent, and may occur after the otitis media has apparently subsided, these cases require careful differential x-rays and thorough and repeated examination. Not infrequently a septum of apparently solid sound bone will be found separating the zygomatico-temporal cells from those about the mastoid antrum. Because such cells may extend far forward and downward, drainage is impeded and healing may be prolonged.

FENTON.

Thrombosis of the Lateral Sinus.

Evans, William H. (Youngstown, Ohio). Arch. Otolaryng., 28:959 (Dec.), 1938.

This article is a survey of the current literature dealing with thrombosis of the lateral sinus. The author has collected a series of 59,850 cases of mastoid disease from approximately 343 physicians and hospitals, in which thrombosis of the lateral sinus occurred in 1,556 cases. These figures show an incidence of 2.6 per cent for thrombosis of the lateral sinus in mastoid disease. There were 305 deaths, or a mortality rate of 31.2 per cent. The incidence seems to be higher in children's and charity hospitals and in private practice largely among referred patients. Climate seems to have no effect upon the incidence of the condition.

Although many authors oppose operative intervention on the jugular vein in treatment of thrombosis of the lateral sinus, this survey shows that exposure of the lateral sinus with ligation of the jugular vein is still the method of choice. This latter procedure, supplemented by transfusions, specific serums, and drugs such as sulfanilamide, is much in vogue at the present time.

The author concludes with a plea to the individual physician and surgeon to make careful observations and keep complete and accurate records. By the use of this collective experience and knowledge only can the individual otologist hope to improve his results.

TOBEY.

MISCELLANEOUS

Lipoid Pneumonia in Infants and Children.

Bromer, Ralph S., and Wolman, Irving J. (Philadelphia). Radiology, 32:1-7, (Jan.), 1939.

Twenty-seven cases of lipoid pneumonia in infants and children are reported. All but four of the children were less than two years old, and two-thirds of the patients were suffering from some debilitating condition.

Chronic cough was present in six cases. Physical examination of the chest was negative or nearly so in all cases.

At autopsy (20 cases), the oil was found to assume a characteristic disposition, namely, in the posterior and dependent portions of the lungs and in the perihilar regions. The right lung was more involved than the left.

The authors believe a roentgenographic diagnosis can be made in severe cases, due to the characteristic location of the lesions. In moderate and mild cases, serial roentgenograms and an accurate history are needed.

They warn against the use of oily nose drops in small or weak children, and against forcing cod-liver oil or liquid petrolatum orally.

DEAN, JR.

Temperature of the Upper and Lower Respiratory Tract. (La temperatura delle vie respiratorie superiori ed inferiori.)

Ricci, C., and Scevola, P. (Milano). Arch. Ital. di Otol., Rino., Laring., 50:1 (January), 1938.

The Benedict-Comel apparatus was employed to register the temperature of the air in the oral cavity, hypopharynx, glottis, upper and lower regions of the trachea and large bronchi, both during inspiration and expiration. Normal individuals from the second to the fifth decade were chosen. Their temperature, pulse and respiratory rate were recorded and likewise the temperature and humidity of the room. A two per cent pantocaine solution was used for anesthesia when indicated, and a special endoscopic instrument had to be constructed to prevent the thermoelectric apparatus from contacting the walls of the respiratory tract.

In one table the authors report the air temperature found in the various regions of the respiratory apparatus of 25 individuals. Here one notes that during inspiration there is a gradual increase in

temperature as the air proceeds from the mouth to the bronchi. The difference in temperature between the air in the oral cavity and the large bronchi is of five degrees centigrade when inspiration takes place through the mouth, and of two degrees centigrade when the air is inhaled through the nose. This marked difference in temperature between the two methods of inhalation, that is, through the mouth or nose, is evidenced only in the oral cavity, while in other regions all the way from the hypopharynx to the large bronchi it varied approximately five-tenths degree centigrade.

Deviations have also been found when the room temperature was lowered, or the depth and rate of respirations were changed and, in some individuals, the anesthesia also had some influence.

During expiration through the mouth or the nose, the difference in the temperature is very negligible except in the oral cavity, where it registered one degree lower when expiration took place through the open mouth.

The second table records the temperature of the mucosa in the various regions of the respiratory tract. The heat of the mucosa is also influenced by the same factors stated in the previous experiment.

The results of their experiment, in general, coincide with those reported by earlier investigators who have used numerous methods for recording these findings.

SCIARRETTA.

Histological Comments on Benign Tumors of the Tongue. (Commento ai reperti istologici di tumori benigni della lingua.)

Bozzi, E. (Milano). Arch. Ital. di Otol., Rino., Laring., 49 (Nov.-Dec.), 1937.

This interesting histological treatise covers almost two issues of the Italian Archives of Otology. The cases were collected at the Victor Emanuel III Institute for the Study and Cure of Cancer in Milan during the years 1928-1937.

The author points out that the rarity of these benign tumors is due to the fact that many of these growths are removed by the general practitioner because of their small and benign appearance; they are considered of secondary importance and are not histologically checked; consequently, they are thought to occur infrequently. Evidently very few reports appear in the literature.

Bozzi, in his work, presents in detail a case history of lipoma, fibroma, papilloma, angioma, fibroangioma, lymphangioma and two of hemolymphangioma. He discusses minutely the histological structure of each neoplasm, the genesis and classification, and the previous works published on each type of growth.

This paper is thorough and should be of importance to the pathologist and otolaryngologist interested in histological study. Many photomicrographs and an extensive international bibliography accompany the article.

SCIARRETTA.

The Upper Air Passages in Pellagra. (De l'état des voies aériennes supérieures chez les malades atteints de pellagre.)

Dorochenko, I. T. (Velikoic, Zaporojic, U. S. S. R.) Acta Oto-Lar., 26:6-658 (Nov.-Dec.), 1938.

Pointing out that lesions of the buccal and pharyngeal mucosa are among the earliest symptoms of pellagra, and that laryngologists frequently confuse these symptoms with ordinary stomatitis, glossitis or Vincent's angina, the author states that symptomatic management is all that the specialist should undertake, keeping the patient in bed, under the care of the internist.

Complaint is made of burning and painful swallowing, involving mouth, tongue and pharynx, with increased salivary secretion. Locally, all cases showed hyperemia of the buccal mucosa; 42 per cent superficial ulcers of the mucosa of the cheeks; 34 per cent softening, ulceration and bleeding of the gums; 62 per cent tonsillar hyperemia; 11 per cent exfoliation or ulceration of epithelium of epiglottis; 20 per cent hyperemia and superficial ulceration of septal mucosa; 11 per cent exposure of vessels in Kiesselbach's area; 12 per cent mucosal changes in upper esophagus; 7 per cent epiglottic hyperemia with edema of true and false cords; 17 per cent changes in the nasal skin and of the folds of the upper lip. Secondary infection, due to enfeebled general resistance, is responsible for the severity of these local symptoms. Tracheotomy was not necessary for any of the cases of laryngeal edema. The laryngologist has a distinct responsibility for recognizing these manifestations, as he is often the first to see such cases.

FENTON.

The Culpability for Delay in the Treatment of Cancer.

Park, George, and Gallo, James (From the Memorial Hospital, New York, and the Josephine Lendrian Tumor Clinic, Paterson General Hospital, Paterson, N. J.), Am. J. Cancer, 32:443 (July), 1938.

An analysis is made of 1000 cases at random, seen during the past ten to fifteen years in these two hospitals, to determine responsibility for delay in the treatment of cancer of all types. Delay of three months is considered undue. The patient alone was responsible for 44.3 per cent of delays, patient and physician 18 per cent, physician alone 17 per cent, no delay 20.7 per cent. Along with a campaign to educate the public, there should be further effort made to train physicians in the matter of early diagnosis in order that patients may receive adequate treatment leading to a greater chance for cure.

JORSTAD.

Bronchoscopy in Pulmonary Tuberculosis.

Morlock, H. V. (London), and Hudson, E. H. (London). Brit. Med. J., 4077:381 (Feb. 25), 1939.

The authors feel that bronchoscopy can be helpful and is indicated in five types of pulmonary tuberculosis, namely, sputum-negative cases; those showing areas of collapse; those showing areas of obstructive emphysema; those with symptoms suggestive of tracheobroncial tuberculosis, and those in which another pulmonary disease is associated with pulmonary tuberculosis.

The signs and symptoms and some illustrative cases of these groups are presented.

DEAN, JR.

Books Received

Loose-Leaf Surgery of the Ear.

Prepared Under the Editorship of Dr. Samuel J. Kopetzky, Professor of Otology, New York Polyclinic Medical School and Hospital; and Attending Otolaryngologist, Beth Israel Hospital, New York. Cloth. Royal 8vo. of 456 pages, illustrated with 292 cuts and four color plates. New York: Thomas Nelson & Sons, 1938. Price, $12.00.

Seventeen authors of distinction have collaborated to produce what constitutes the definitive edition of ear surgery as it is practiced today. Not content with this, they have bound it loose-leaf so that it will have every chance of remaining such with the changing years.

The surgery of the ear has enjoyed unusual attention, and hence unusual advancement in the past decade, and it is an almost bewildering literature which confronts the reader. Selection, too, is difficult. It is a field of surgery in which errors are apt to be disastrous, and the young surgeon cannot afford for long to follow false prophets.

A ripe experience lies behind this collection of essays. The compilers have rendered a distinguished service to American otology.

A Manual of Reparative Plastic Surgery.

Dr. J. Eastman Sheehan, Professor of Plastic Reparative Surgery, New York Polyclinic Medical School and Hospital; Surgeon, St. Clare's Hospital; Consulting Surgeon, Morrisania City Hospital, New York; Honorary Professor de la Academia de Sanidad Militar, Spain. Cloth. 8vo. of 311 pages with 314 illustrations and 18 full-page plates. New York: Paul B. Hoeber, Inc., 1938. Price, $5.50.

Dating back to the Italian Renaissance, the art of reconstructive surgery has progressed with the great wars. Culminating in the unprecedented experiences of the World War, unprecedented because of their number and the advanced knowledge of anesthesia and asepsis, this branch of surgery has developed new knowledge regarding the serviceability of the various tissues in reconstruction, the behavior of muscles, bone, skin, nerve and tendon in new positions and environments, and an instrumentarium designed to cope with its special problems.

The first part of the present volume deals with these fundamentals—the tools and materials.

The second part deals with specific operations. The author describes those procedures which have given him the best results, avoiding any academic enumeration of known procedures.

The book is essentially a surgical manual, rich in detail both textual and graphic.

A Treatise on the Surgical Technique of Otorhinolaryngology.

Georges Portmann, Professor of Otorhinolaryngology at the Medical School of the University of Bordeaux, and Collaborators. Translated by Pierre Viole, M.D., Associate Clinical Professor of Surgery in the Department of Otorhinolaryngology of the University of Southern California School of Medicine. Cloth. Royal 8vo of 675 pages with 474 illustrations, 2 color plates. Baltimore: Williams & Wilkins Company, 1939. Price, $12.50.

This manual describes in minute and vivid detail the operative procedures of otolaryngology as practiced by the author. The presentation is terse and methodical. Each operation is discussed under the following headings: indications and contra-indications, preparation of the patient, anesthesia, instruments, technique and post-operative treatment, including the management of complications. The technique is divided into steps, and each step is illustrated with a life-sized drawing.

These drawings are rather remarkable for their diagrammatic clarity and the fact that they are so turned on the page that the reader sees them from the same angle as he will approach his patient on the operating table. All instruments are also illustrated.

The translation is excellent, and the translator deserves great commendation for the fidelity with which he has maintained the spirit of the original.

The author has departed from the usual textbook method of presenting all accepted methods impartially, leaving the reader unadvised, to make his own choice. Accordingly, there are occasional passages in the book which are at variance with the more commonly accepted American teachings of the moment; however, they represent the ripe experience of a large and well conducted clinic and are entitled to thoughtful attention.

Diseases of the Ear, Nose and Throat.

Francis L. Lederer, B.Sc., M.D., F.A.C.S., Professor and Head of the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, Chicago; Chief of the Otolaryngological Service, Research and Educational Hospital. Cloth. Royal 8vo. of 835 pages, illustrated with nearly 500 halftone and line engravings, mostly original, and 16 full-page color plates. Philadelphia: F. A. Davis Company, 1938. Price, $10.00.

This general textbook of otolaryngology is rather more elaborate in its format than most. Owing to the large page-size, the material is set in double column which, together with a clear, legible type face, makes for easy reading.

It has been the author's special endeavor to include everything of moment, in which he has succeeded admirably. To accomplish this, however, he has been obliged to omit a certain amount of elaboration of detail which would be welcomed by the teacher and the specialist. The trained otolaryngologist today is demanding to know more and more about the minutiae of his specialty. Obviously a textbook in one volume cannot supply these, but one feels that it should contain extensive references and should quote its sources if it is to appeal greatly to the serious graduate student and the teacher, whose concepts may not be in agreement with those laid down by the author.

The illustrations are definitely of a type which will engage the attention of the advanced student. They are exceptionally clear and well chosen, and there is a wealth of pathological material both microscopic and gross. There are clinical photographs of almost every condition capable of depiction, chiefly of the advanced stages, and in such profusion as to give the book almost the character of an atlas.

Five monographs of exceptional interest issued from the press of the Acta Oto-Laryngologia during the year just past:

Bakteriologisch-Klinische Untersuchungen Über Operativ Behandelte Komplikationen der Akuten Mittelohrentzündung (Bacteriological-Clinical Studies of the Surgical Complications of Acute Otitis Media).

Eino Vaheri, from the Otolaryngological Clinic of the University of Helsinki. Paper. Supplementum XXIX of Acta Oto-Laryngologica, 227 pages. Helsinki, 1938. (In German.)

Pathologisch-Anatomische Studien Über die Kehlkopftuberkulose (Pathologic-Anatomic Studies of Laryngeal Tuberculosis).

Torsten Blomroos, from the Tuberculosis Hospital in Helsingfors. Paper. Supplementum XXVIII of Acta Oto-Laryngologica, 156 pages with 39 illustrations. Helsingfors, 1938. (In German.)

Das Blutbild und die Senkungsreaktion bei Otogenen Infektionen (Blood Picture and Precipitation Tests in Otogenic Infections).

Paavo Koskinen, from the Otolaryngological Clinic of the University of Helsinki. Paper. Supplementum XXV of Acta Oto-Laryngologica, 268 pages. Helsingfors, 1938. (In German.)

Pathologie und Klinik der Altersschwerhörigkeit (Clinical Pathology of Senile Deafness).

Arno Saxen, Helsingfors, Finland. Paper. Supplementum XXIII of Acta Oto-Laryngologica, 101 pages with 33 illustrations. Helsingfors, 1937. (In German.)

A Contribution to the Physiology of Bone Conduction.

Ernst Bárány, from the Oto-Rhino-Laryngological Clinic of the Royal Academic Hospital, Upsala, Sweden. Paper. Supplementum XXVI of Acta Oto-Laryngologica. Uppsala, 1938. (In English.)

ANTONIE P. VOISLAWSKY

1872-1939

Doctor Antonie P. Voislawsky of New York City died on February 22nd, 1939.

Doctor Voislawsky was born in New York City June 5th, 1872. He received his education from the New York University. After his graduation in 1894, he entered Dartmouth College, receiving his medical degree in 1897.

He was a Fellow of the American Laryngological, Rhinological and Otological Society; American Medical Association; American College of Surgeons; American Otological Society and the New York Laryngological Society.

He was Director of the Nose and Throat Department, Laryngologist, St. Luke's Hospital; Attending Otolaryngologist, Staten Island Hospital, Tompkinsville; Consulting Otologist at the Harlem Eye, Ear and Throat Infirmary, Manhattan Maternity and Dispensary, New York and the Northern Westchester Hospital, Mt. Kisco; Consulting Laryngologist, New York Society for the Relief of the Ruptured and Crippled; Laryngologist, Fifth Avenue Hospital, New York.

He was the author of numerous articles, but most of his contributions appeared in the earlier years of his professional career.

In 1902 he married Margaret R. Van Rensselaer, who survives him. He leaves also a son, Van Rensselaer, who was associated with him in the practice of his specialty, and a daughter, Elizabeth Van Rensselaer.

PATRICK WATSON-WILLIAMS

1860-1938

Patrick Watson-Williams, Honorary Consulting Surgeon in Diseases of the Ear, Nose and Throat at the Bristol Royal Infirmary, and one of the best known English otolaryngologists, died on November 18th at the age of seventy-eight.

For many years his name was prominent in the British publications and his influence upon the surgical aspects of nasal treatment was widespread. Much of his work is epitomized in his monograph "Chronic Nasal Sinusitis and Its Relation to General Medicine," published in 1930.

In association with Graves and Pickworth, he made valuable contributions to the knowledge of mental disease in relationship to infection of the nasal accessory sinuses.

He was onetime Lecturer on Oto-Rhino-Laryngology in the University of Bristol; Honorary Member of the Laryngological Society of Vienna and Corresponding Member of the Oto-Rhino-Laryngological Society of France.

Notices

Fourth International Congress of Oto-Rhino-Laryngology
Amsterdam, July 29 to August 3, 1940

The Fourth International Congress of Oto-Rhino-Laryngology will take place at Amsterdam from July 29 to August 3, 1940.

HONORARY CHAIRMEN

Dr. H. Colijn, Prime Minister.

Mr. J. A. N. Patijn, Minister of Foreign Affairs.

Prof. Dr. J. R. Slotemaker de Bruïne, Minister of Education, Arts and Sciences.

Prof. Mr. C. P. M. Romme, Minister of Social Affairs.

ORGANIZING COMMITTEE

Prof. Dr. H. Burger, President.

Dr. A. Marres, First Secretary, Willemsparkweg 31 Amsterdam.

Dr. A. J. H. Dokkum, Joint Secretary,

Dr. M. J. ten Cate, Treasurer.

The three official subjects for discussion before this Congress will be:

1. "Ciliary Movement of Mucous Membranes of the Air Passages." Reporters—Prof. Dr. A. Hermann, Erfurt (Germany): Ciliary Movement of the Mucous Membranes. Mr. V. E. Negus, London (England): Ciliary Movement in the Larynx and Lower Air Passages. Dr. Arthur W. Proetz, St. Louis, Mo. (U. S. A.): Ciliary Action in the Nose.

II. "Allergic Diseases." Reporters—Dr. Paul Kallós, Stockholm, (Sweden): Allergy from a General Point of View. Prof. C. E. Benjamins, Groningen (Holland): Hay Fever. Dr. French K. Hansel, St. Louis, Mo. (U. S. A.): Allergy in Otolaryngology and Its Relation to Other Manifestations.

III. "Ménière's Disease." Reporters—Dr. Maurice Aubry and Dr. Marcel Ombridanne, Paris (France): Surgical Treatment of Ménière's Disease. Prof. Dr. Giorgio Ferreri, Rome (Italy): Non-Surgical Treatment of Ménière's Disease.

Graduate Course in Otolaryngology of Washington University

The graduate course in otolaryngology at Washington University for 1939-1940 will begin on September 25, 1939. Information may be had by addressing the Department of Otolaryngology or Mr. William B. Parker, Registrar, Washington University School of Medicine, Scott and Euclid Avenues, St. Louis.

University of Cincinnati Course in Surgical Anatomy May 29 to June 3, 1939

The third annual course in otolaryngology will be given May 29 to June 3, 1939, inclusive, by the Departments of Otolaryngology and Anatomy in the Medical College of the University of Cincinnati. This is an intensive operative course on the cadaver, and abundant material will be available for the standard operations on the middle ear, mastoid, accessory sinuses and the larynx.

Past Issues Wanted

The management of the Annals desires to secure a few copies of the issues of March, 1937, and March, 1938, which are out of print. They will pay $1.00 for them.

There is also an inquiry for Volumes 1 to 30 complete and we would like to communicate with anyone who has this series of which he would care to dispose. Address the Annals of Otology, Rhinology and Laryngology, 7200 Wydown Boulevard, St. Louis.

Communication from the Balkan Medical Union

The following communication from the Balkan Medical Union is of interest to laryngologists who may be concerned at any time with the ravages of gas warfare. It is a sign of the times and a significant sidelight on the Balkan viewpoint at the moment.

The Union was formed in 1931 for the purpose of engendering and maintaining mutual understanding among the scientific men of the nations.

At Istanbul, December 31st, 1938, the following resolutions were adopted:

The Balkan Medical Union, in session at Istanbul,

having taken into consideration the terrible sufferings which a total war will bring upon the civil population of open towns together with the total lack of any adequate means of protection,

and having discovered that even in its restricted form the project of "sanitary towns" has not yet been adopted, and that all efforts made to protect civilians against chemical warfare have till now remained as proposals only, and that even the protocol prohibiting the use of asphyxiating gas has not yet been ratified by all nations,

has therefore decided to address itself to doctors of every nation with an appeal to take active measures and to fulfill this professional and humanitarian duty of awakening and stirring public opinion . . .

Prof. Dr. Bensis, Dr. Scaramanga (Athènes); Dr. Zika Markoviç, Prof. Dr. K. Sahoviç, Dr. M. Simoviç (Beograd); Prof. Dr. Gheorghiu, Dr. Popescu Buzeu (Bucarest); Prof. Dr. Akil Muhtar Özden, Prof. Sedat Tavat, Prof. Dr. A. Süheyl Ünver (Istanbul).

This communication was "addressed to the medical journals of the whole world with the aim of arousing if possible a favorable public opinion for adopting the necessary measures against the dangers to which the civil population will be exposed during a 'total war'."

Notices of Coming Meetings

American Laryngological, Rhinological and Otological Society, Chicago, Illinois, May 9 to May 11.

American Medical Association, St. Louis, Missouri, May 15 to May 19.

American Otological Society, Rye, New York, May 22 to May 23.

American Laryngological Association, Rye, New York, May 24 to May 26.

American Bronchoscopic Society, Rye, New York, May 26.